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OPERATIVE ORAL SURGERY

PLASMACYTOMA OF MANDIBLE

Report of a Case

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PLASMACYTOMA or solitary myeloma is a single focus of myelomatous tissue, without dissemination to other parts of the skeleton. In the majority of the reported cases of plasmacytoma, the follow-up data were insufficient for adequate evaluation of subsequent dissemination; thus, the existence of such a solitary lesion remains a controversial issue. The following are essential for the diagnosis of plasmacytoma: (1) establishment of the lesion's identity by biopsy; (2) the elimination of the presence of other such lesions by a roentgenographic skeletal survey; (3) a negative report on the bone marrow study and, as a rule, (4) absence of hyperglobulinemia and Bence Jones proteinuria. However, slight variations in the electrophoretic pattern of the blood may be present.

CASE REPORT

History.—On Aug. 6, 1956, a 64-year-old Negro man was admitted to the Veterans Administration Hospital in Tuskegee, Alabama. The following diseases had been diagnosed: chronic, simple glaucoma, hypertensive cardiovascular disease, and generalized arteriosclerosis. During a routine dental examination, periapical roentgenograms revealed a circumscribed roentgenolucent area in the left mandibular molar region (Fig. 1) which extended beyond the edge of the film. The patient was not aware of the lesion's existence.

The patient had been hospitalized in 1950 for treatment of a urethral stricture, cardiac dilatation, and hypertrophy. An erythrocyte count of 4,000,000 per cubic millimeter, a hemoglobin value of 11 Gm., and 1+ albumin were the only significant laboratory findings.

Clinical Examination.—Except for a periodontal condition and the absence of several teeth, the intraoral and extraoral findings were essentially negative.

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Fig. 1.—Periapical roentgenogram of left mandibular molar region.

Fig. 2.—Posteroanterior view showing multilocular roentgenolucent lesion of left mandible.

Roentgenographic Examination.—Periapical roentgenograms, posteroanterior (Fig. 2) and left lateral (Fig. 3) films of the mandible, and views of the skull, chest, pelvis, and extremities were taken.

The x-ray examination revealed a multilocular roentgenolucent area in the body of the left mandible. The area extended posteriorly from the second premolar to within approximately 3 cm. of the angle and inferiorly from the superior border to within 1 cm. of the inferior border of the body of the mandible. A line of increased roentgenopacity surrounded the area of roentgenolucency. There was no other roentgenographic evidence of bone pathology.

Fig. 3.—Left lateral view of mandible.

Laboratory Examination.—Laboratory findings were as follows:

Hemogram

Erythrocytes, 4,280,000 per cubic millimeter
Neutrophils, 53 per cent
Lymphocytes, 41 per cent

Blood Chemistry

Oct. 22, 1956: Total protein, 6.9 Gm. per 100 c.c. (albumin, 4.0 Gm.; globulin, 2.9 Gm.)
Oct. 29, 1956: Total protein, 8.0 Gm. per 100 c.c. (albumin, 3.6 Gm.; globulin, 4.4 Gm.)
Calcium, 10.4 mg. per 100 c.c.
Inorganic phosphorus, 3.2 mg. per 100 c.c.
Acid phosphatase, 0.8 unit
Alkaline phosphatase, 1.3 units

Urinalysis

Specific gravity, 1.020
Reaction, acid
Sugar, negative
Albumin, negative
Bence Jones protein, negative
Bone marrow studies—Essentially negative.

Tentative Diagnoses.—On the basis of clinical roentgenographic, and laboratory findings tentative diagnoses of ameloblastoma, multilocular cyst, and giant-cell tumor of the body of the left mandible were returned.

Biopsy.—Anesthesia was obtained by blocking of the left inferior alveolar and lingual nerves and by infiltration of the buccal nerve with 2 per cent Xylocaine—epinephrine solution. A mucoperiosteal flap was elevated over the lesion. In the retromolar region the superior border of the mandible was found to be replaced with soft tissue; a section of the tissue was removed. The biopsy report indicated plasmocytoma.

Operation.—The patient was placed under endotracheal anesthesia with thiopental sodium and nitrous oxide and oxygen, and the lesion of the mandible was excised.

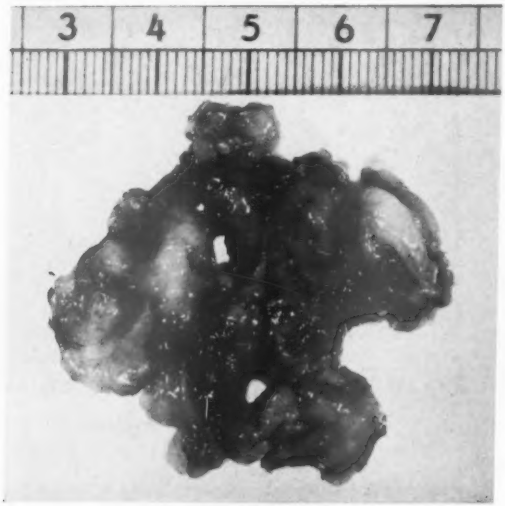

Fig. 4.—Gross specimen.

An intraoral incision was started slightly below the left coronoid process and extended inferiorly along the ramus to the body of the mandible and around the crevices of the teeth to the interproximal space between the first and second left premolars, where it was terminated in the mucobuccal fornix. The soft tissues were reflected, the overlying bone was removed with bone chisels and rongeurs, and the neoplastic tissue was enucleated. The remaining cavity in the bone had smooth surfaces and showed no clinical evidence of infiltration. The bone edges were smoothed, the defect was irrigated with normal saline solution, and Gelfoam saturated with aqueous penicillin was inserted. The soft tissues were approximated and sutured.

Pathologic Findings.—Gross description: The gross specimen consisted of several irregularly shaped masses of brownish tissue which varied from 5 mm. to 2 cm. in diameter (Fig. 4), sections of bone, and a segment of tissue which grossly resembled some type of membrane.

Microscopic description: Sections of the specimen showed a cellular tumor in which the cells appeared to be predominantly mature plasma cells. In the active area of the tumor the cells were closely packed; peripherally, they were dispersed in a hyalinized homogenous stroma (Fig. 5).

Diagnosis: Plasmacytoma of the left mandible.

Follow-up.—On April 17, 1957, six months after the plasmacytoma was excised, the patient was hospitalized for evaluation. The clinical examination revealed no significant findings.

The laboratory studies were as follows:

Bence Jones protein, negative
Total protein, 6.2 Gm. per 100 c.c. of blood (albumin, 2.7 Gm.; globulin, 3.5 Gm.)
Electrophoretic study
Normal: Albumin, 69; alpha (1), 7; beta, 9; gamma, 12 per cent; total protein, 7.3 Gm. per cent
Change: Albumin, 60-77; alpha (1), 1-5; alpha (2), 4-10; beta, 5-3; gamma, 7-17 per cent; total protein, 5.9-8.9 Gm. per cent
Bone marrow study, essentially negative.

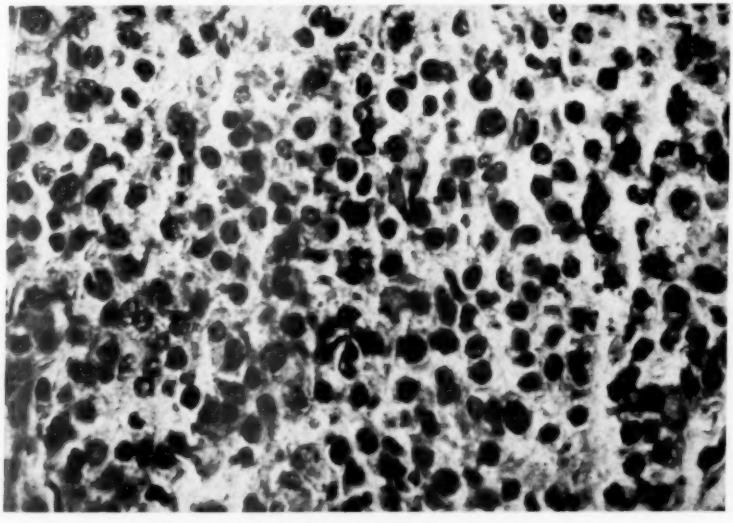

Fig. 5.—Microscopic view of plasmacytoma.

A roentgenographic examination showed the surgical defect of the left mandible. There was no evidence of metastatic lesions in the skeletal system.

On Sept. 19, 1957, the patient was admitted to the hospital with a tentative diagnosis of aneurysm of the aorta.

Laboratory findings were as follows:

Leukocyte count, 9,550 per cubic millimeter
Erythrocyte count, 3,510,000 per cubic millimeter
Hemoglobin, 10.2 Gm.
Calcium, 9.8 mg.
Inorganic phophorus, 4.1 mg.
Alkaline phosphatase, 0.7 units
Total protein, 7.2 Gm. per 100 c.c. of blood (albumin, 3.2 Gm.; globulin, 40 Gm.)
Bence Jones protein, negative.

A hematologist reported that an electrophoretic study (Fig. 6) of the patient's blood showed that the gamma globulin was elevated to twice the normal value. However, there was no evidence of discrete peaks, such as those seen in the blood of patients with cryoglobulinemia or macroglobulinemia.

The roentgenographic survey revealed no skeletal changes indicative of multiple myeloma.

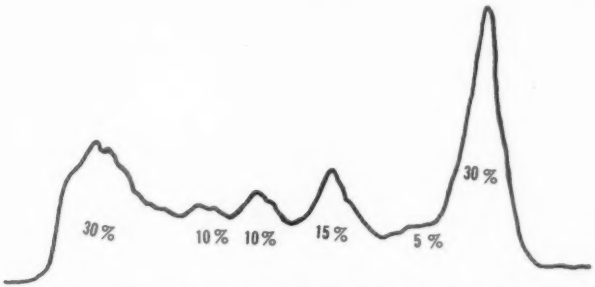

Fig. 6.—Electrophoretic graph of patient's blood during September, 1957.

During May, 1959, the patient was hospitalized for the correction of a cataract. Laboratory studies made to detect possible multiple myeloma were reported as follows:

Leukocyte count, 8,130
Erythrocyte count, 3,400,000 per cubic millimeter
Hemoglobin, 9.85 Gm.
Calcium 9.8 mg.
Inorganic phosphorus, 3.7 mg.
Alkaline phosphatase, 1 unit
Total protein 5.9 Gm. per 100 c.c. of blood (albumin, 3.1 Gm.; globulin, 2.8 Gm.)
Bence Jones protein, negative
Bone marrow study, negative

The roentgenographic survey revealed no skeletal changes indicative of multiple myeloma, and the electrophoretic study did not give readings suggestive of cryoglobulinemia or macroglobulinemia.

COMMENT

The case of a patient with a single focus of myelomatous tissue in the left mandible has been presented. Since August, 1956, the patient was hospitalized three times. During each hospitalization the patient received a complete examination which included laboratory studies and a roentgenographic skeletal survey; the findings did not justify a diagnosis of multiple myeloma.

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PIRIFORM RIM FIXATION FOR MAXILLARY SULCUS EXTENSION

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IN SULCUS-EXTENSION operations, fixation of the mobilized tissue flap in a new position is an important step in preserving the increase in sulcus depth. In these operations, sutures, tubes, or dentures have been used separately or in combination to fix the flap in position.

Tubes made of rubber or polyethylene are commonly used and are usually held in place by sutures extending from the tube and flap through the lip or cheek. Sometimes sutures only are passed from the flap through the lip or cheek. However, tubes and sutures placed in this fashion are subject to movement caused by contraction of facial muscles, and as the area operated upon is usually tender, muscle and skin movement causes some postoperative discomfort. In addition, the direction of fixation from flap to skin is obliquely away from the ridge, and this is usually undesirable.

Fixation of the sutures to the tube from the piriform rim was tried in an effort to improve stability of the tube, to reduce soreness, and to make the suture pull the tube more directly toward the ridge.

The term "piriform rim" as used here refers to the rim of bone on the periphery of the piriform aperture,[1, 2] which is the anterior nasal opening and is bounded bilaterally by the nasal and maxillary bones and split by the septal cartilage. The piriform aperture has also been called the anterior nasal aperture.[2, 3] The rim of maxillary bone on the lateral part of the aperture was that part of the piriform rim used for fixation purposes. The use of this rim for intermaxillary fixation in edentulous jaw fractures has been previously described by Thoma.[4]

MATERIALS

Sutures of 00 size are satisfactory for fixing a tube to the piriform rim. Polyethylene intravenous tubing or size 16 French rubber catheters are adequate for tubes.

TECHNIQUE

Piriform rim fixation can be used for different methods of sulcus extension, as it is suitable for maxillary fixation of sutures, tubes, or dentures. How-

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ever, supplementary means of fixation may be required. The use of the piriform rim for tube fixation in a modification of the Clark sulcus-extension procedure[5] will be described.

The gingiva is incised and the depth of the sulcus is increased by supraperiosteal dissection (Fig. 1). The mucosa is then undermined by blunt dissection and mobilized into its new position.

Fig. 1.—Supraperiosteal and submucosal incisions in maxilla.

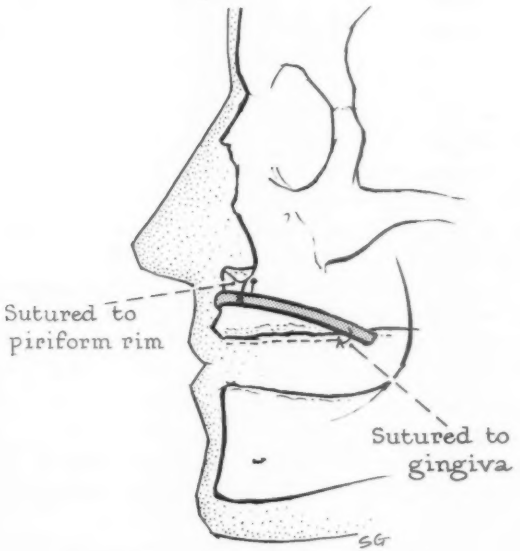

Fig. 2.—Hole drilled in lateral piriform rim. Tube sutured to rim and gingiva.

If the nasal spine is prominent, it is reduced in size after a periosteal flap has been raised. The periosteum may have to be resutured over the reduced nasal spine.

The piriform rim is palpated and an incision is made in the periosteum just below the rim. A periosteal flap is then raised for exposure of the rim. In addition, the nasal mucoperiosteal flap is raised from the internal side of the rim. This exposes the rim completely, and a hole is drilled from without inward (Fig. 2). The hole is made on the lateral side of the rim, as the rim

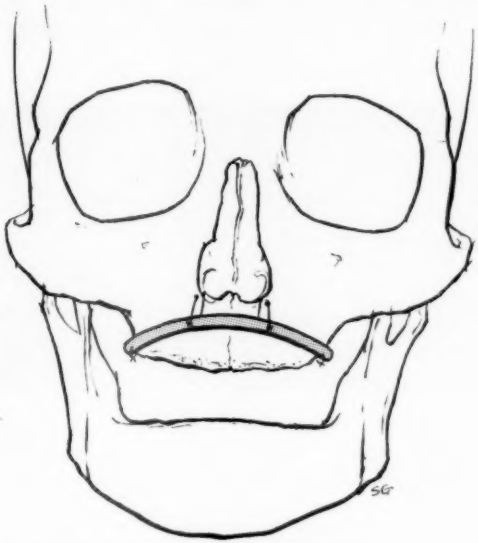

Fig. 3.—Bilateral holes in piriform rim.

Fig. 4.—Tube sutured through edge of flap to piriform rim. Periosteum exposed and flap in new position.

is usually sharp there and composed primarily of cortical bone. The inferior side of the rim is avoided, as it is usually flat. Holes are drilled bilaterally (Fig. 3).

The suture is placed through the edge of the flap, passed through the hole, and brought through the edge of the flap again (Fig. 4). At this stage,

the edges of the mucosal flap may be sutured to the periosteum before the tube is tied in place. This supplements the tube in fixing the flap. The suture is then placed around the tube and tied. Usually the tube is also sutured to the attached gingiva or mucosa near each end of the tube for additional stability. The tube is thus fixed by at least four sutures. The ends of the tube are left slightly long and are directed away from the sulcus so as not to dig into the soft tissue. The periosteum-covered surface then granulates and epithelizes

Fig. 5.

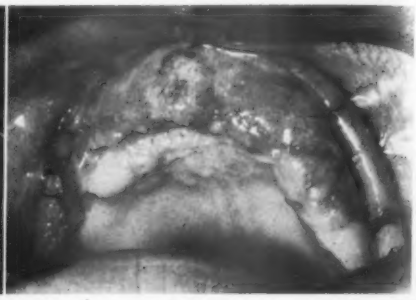

Fig. 6.

Fig. 5.—Shallow labial sulcus of maxilla shown preoperatively. Note slight amount of hyperplastic tissue on ridge crest.
Fig. 6.—Seven days postoperatively.

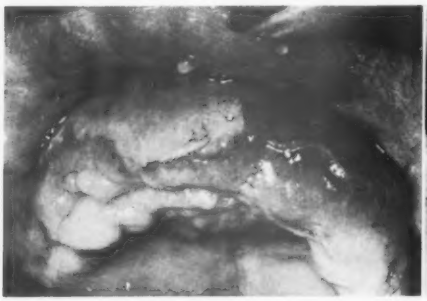

Fig. 7.

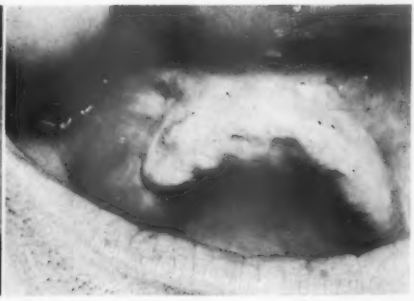

Fig. 8.

Fig. 7.—Ten days postoperatively, after removal of tube.
Fig. 8.—Fourteen months postoperatively.

(A shallow maxillary sulcus before operation is shown in Fig. 5.) The tube is removed after about ten days (Fig. 6). As soon as possible after removal of the tube, a denture is placed to maintain the increase in sulcus size which has been obtained surgically (Fig. 7). Some loss of sulcus depth due to scar contraction can be expected (Fig. 8).

DISCUSSION

Healing time has been similar with this and other methods of sulcus extension. Less soreness in the cheek and lip has been noted after piriform rim fixation than following the use of through-and-through skin sutures.

The duration and extent of the surgical procedure are about the same when the piriform rim is used for fixation as when through-and-through skin sutures or dentures are used.

Flap fixation was more stable, as the flap was fixed to bone rather than to movable skin and muscle. The fact that the tube was not moved by skin and muscle movements explained why there was less soreness.

With the piriform rim technique the sutures around the tube pull the flap toward the ridge, whereas skin sutures fix the flap in a direction away from the ridge. It is more desirable to maintain the edge of the flap close to the ridge in the Clark procedure in order to reduce formation of scar tissue in the depth of the sulcus.

Esthetically, the piriform rim fixation was better because the external skin sutures and bandages required by some techniques were not necessary.

Other basic methods of sulcus extension[6-8] appear to be adaptable to piriform rim fixation with modifications. The modified Clark method was used here solely to illustrate the piriform rim fixation technique. No comparative advantages over other sulcus-extension techniques was intended or considered in this report, as this is a separate subject of investigation in itself.

SUMMARY

The piriform rim can be used for fixation of tubes in sulcus-extension procedures. The technique has been described. The use of the piriform rim for fixation of sutures or dentures and for use in other sulcus-extension techniques appears possible and requires further investigation.

Fixation to the piriform rim was more stable than the use of through-and-through skin sutures. The tube and flap were pulled toward the ridge in the area of fixation.

Because the tube was fixed to bone, postoperative soreness resulting from movement of skin sutures was not possible. Esthetically, the internal piriform rim fixation technique is preferable because it does not require the external skin sutures used in some techniques.

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CONTROL OF BLEEDING WITH PREMARIN INTRAVENOUS IN ORAL SURGERY

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EVEN though excessive bleeding and the threat of exsanguination do not constitute in dentistry the danger that they do in other branches of surgery, Johnson[1] has demonstrated that "the extent of hemorrhage in oral surgery has been vastly underestimated." The danger of postoperative hemorrhage is always present, and it is further complicated by its unpredictability. Routine bleeding, clotting, prothrombin, and clot retraction times can be useful in revealing a potential bleeding tendency, but probably the most important factor is the patient's history, familial as well as personal. McIlvanie,[2] reporting on twenty-four patients with thrombopathic states characterized by a congenital prolonged bleeding time, noted that the most important finding was a history of excessive bleeding following tooth extractions. Furthermore, these patients often bleed more after minor procedures than they do after major operations.

It must be remembered, however, that known bleeders may lose only a minimal amount of blood, whereas patients with a negative history and no laboratory evidence of an abnormal hemostatic mechanism can bleed profusely. These latter patients constitute a majority of the bleeders encountered.

Cheraskin[3] believes that the most common causes of excessive bleeding in oral surgery are (1) defects in the vessel wall (vascular), (2) abnormalities of the tissues surrounding the vascular bed (extracellular), and (3) hemorrhagic disorders (intravascular). Also, changes in tissue tone, hyperemia, and the presence of granulation tissue increase the bleeding potential.[1]

Whatever the cause, however, such bleeding can be a serious complication that is difficult to control, even with hospital care and heroic emergency measures. Furthermore, therapy must be directed not only at stopping the bleeding but also at facilitation of healing, since hemorrhage is likely to recur until healing is completed.

A prophylactic styptic measure, therefore, seems indicated for all oral surgery patients and manditory for patients suspected of being or known to be bleeders. This article presents an evaluation of the use of Premarin intravenous as a routine preoperative measure to reduce bleeding during and after surgery.

Premarin intravenous is the only estrogenic preparation suitable for administration by vein. It is an aqueous solution of soluble conjugated estrogens

Premarin intravenous is a product of Ayerst Laboratories, New York, New York.
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derived from the urine of pregnant mares. It contains, in addition to sodium estrone sulfate, the sulfates of equilin, equilenin, β-estradiol, and β-dihydroequilenin. Other α- and β-estrogenic diols are also present in varying amounts as water-soluable conjugates. When reconstituted with the 5 c.c. diluent, a vial of Premarin intravenous contains 4 mg. of estrogen per cubic centimeter.

As an estrogen, the drug has a direct influence on sex-linked tissues—the endometrium, the ovaries, the vaginal mucosa, the breasts, and the prostate gland. It has now been pointed out that in addition to these effects as a "sex steroid," it has many significant metabolic actions. Estrogens increase osteoblastic activity; they have minor but beneficial effects on electrolyte balance and nitrogen retention; they increase the hydration of the dermis and stimulate the elastic fibrils. Evidence is also accumulating that estrogens offer some protection against atherosclerosis.

These various actions will not be noticed with Premarin intravenous therapy, however, unless the drug is given frequently and over an extended period of time. When Premarin intravenous is used as a physiologic hemostat, usually no more than three or four injections are given, and this is not sufficient estrogenic stimulation to produce the other actions. Therefore, the possibility of undesirable side effects is negligible.

Beginning with Jacobson's[4, 5] description of spontaneous bleeding (sudden, unexpected oozing from a mucosal surface), which seems to describe many bleeding episodes in oral surgery, reports in the medical literature have described the efficacy and safety of conjugated estrogens equine as a parenteral hemostat. The mechanism of action has not been fully explained, but it may be due to a direct effect on the clotting mechanism and/or the extravascular tissues.

The use of this agent in dentistry was first reported by Whittington,[5] who found that one 20 mg. injection controlled a bleeding episode, with no recurrence, in a patient who bled profusely following a routine extraction and failed to respond to all other therapy. Young[6] reported on the use of Premarin intravenous to control massive oral hemorrhage in patients under general anesthesia. In nineteen out of twenty patients the operative field cleared within five minutes after administration; there were no side effects during or following surgical procedures.

OUTLINE OF STUDY

The 1,183 patients reported here were seen in our private oral surgery practice in 1957 and 1958. These patients underwent procedures which included multiple and full-mouth extractions, removal of impactions, treatment of maxillofacial traumatic injuries, excision of massive malignant growths, antral surgery, and surgical treatment of septic conditions. During the first year one-half of the patients, selected at random, were given preoperative injections of Premarin intravenous. After that Premarin intravenous was used with increasing frequency, and now its use is routine.

Prior to the operation an infusion of 5 per cent dextrose in water was started. After the administration of Pentothal sodium by means of a needle

with a two-way stopcock, the intravenous estrogen was injected through the same needle. Routinely, patients received one reconstituted vial (20 mg.); those with histories of prolonged bleeding after prior surgical procedures were given two vials (40 mg.). The first patients in this series received Premarin intravenous injections at the time of the first incision. No hemostatic effect was usually noted in the first fifteen minutes, however, and the estrogen preparation is now given shortly before the first incision is made.

Charts were kept for each patient, and the degree of operative and postoperative bleeding was recorded. Routine blood counts were made and bleeding and coagulation times were determined for all patients preoperatively and repeated for 250 patients, selected at random, from one to five hours postoperatively.

RESULTS

In view of the many variables that can affect such an evaluation, the over-all results of the estrogen premedication in this study can be assessed as most satisfactory. In a series of this size, troublesome bleeding would be expected to occur in 10 per cent of the patients during the operation and in 15 per cent afterward. Instead, only one out of 1,183 patients had excessive bleeding, and this was controlled in thirteen minutes with an additional injection at the completion of the operation. No hemostatic other that Premarin intravenous was given any patient.

Probably the most impressive results were seen in patients who had bled excessively following previous operative procedures. When given intravenous estrogen premedication, none of these patients experienced postoperative bleeding.

The effect of Premarin intravenous was usually evident about fifteen minutes after the injection, when a marked decrease in both bony and soft-tissue bleeding could be noted. In the majority of patients, bleeding was minimal during surgical procedures. This hemostatic effect was demonstrated in full-mouth extractions. When the maxillary teeth were removed first, there was the usual amount of bleeding, but by the time the mandibular teeth were removed a definite decrease in bleeding was noted; this was conversely true when the mandibular teeth were extracted first. (For example, in a patient who underwent full-mouth extraction with alveoloplasty, the maxillary teeth were removed first and the flaps were left open. There was the usual amount of bleeding. The mandibular teeth were then extracted and the flaps were sutured. When the maxillary wound was re-examined there was practically no oozing.) In most patients the operative field was completely dry at the end of the operation. There was not a single incident of abnormal postoperative bleeding.

The hemostatic effect of Premarin intravenous was further demonstrated by the decreased number of sutures necessary. For example, only eight sutures were used for the maxillary wounds and six for the mandibular wounds in a patient who had twenty-eight teeth removed. In another patient with four bony

impactions, no suturing was necessary until the extractions had been completed, at which time all areas were dry.

In patients undergoing reduction of the zygomatic arch, there was no evidence of postoperative intraoral hematoma or ecchymosis. Pressure dressings were unnecessary.

The effect of Premarin intravenous was most marked in operations on soft-tissue structures, such as the tongue and the floor of the mouth, and in extraoral procedures.

COMMENTS AND SUMMARY

Some bleeding must be expected in any surgical procedure. The object is to keep it minimal. Probably the most important measure is careful surgical technique. Even with the most meticulous dissection, however, excessive bleeding is encountered in some patients. This study was undertaken in the hope of preventing this unpredictable problem. It is felt that the results (only one incidence of excessive bleeding in 1,183 patients) demonstrate the value of injecting Premarin intravenous as a routine prophylactic measure for hemostasis.

To determine whether or not Premarin intravenous affected the routine preoperative blood tests, these tests were repeated one to five hours after surgery, the time when the clinical effect was noted; there were no significant changes.

No patient had an untoward reaction to the medication.

For all practical purposes, I feel that careful surgery and the use of Premarin intravenous have eliminated the problem of excessive bleeding during and after oral surgery.

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ORAL MEDICINE

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DENTOMEDICAL IMPLICATIONS OF THE RESIDUAL EFFECTS OF BULBAR POLIOMYELITIS

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POLIOMYELITIS is not a new disease, for it was depicted by Egyptian sculptors about 1,500 B.C.[1] Yet, the first mention of this disease in modern medical literature dates back only to 1836.[2] Since then, the world-wide spread, the fatalities, and the maiming effects of poliomyelitis have become matters of great concern to the medical profession. This article will focus on the implications of poliomyelitis which concern the dental profession, and it will suggest ways by which we might effectively contribute to the teamwork against this disease.

Bulbar poliomyelitis, the most serious form, is of particular interest to the dental profession, since it may affect the fifth, seventh, tenth, eleventh, and twelfth cranial nerves. One would expect a fair percentage of the many thousands of polio victims to come to the attention of the dental profession throughout the civilized world. However, a search of the available literature discloses only fourteen reported cases.[3-7] It would be nice if we could accept this insignificant number as proof of the disease's infrequency, on a global basis, over a period of about twenty-five years! The fact that so few cases have been reported may be explained in the light of the following considerations:

1. The polio virus does not frequently affect the nerves of special interest to the dental profession.
2. The severity of many cases of bulbar poliomyelitis may cause the attending physician to concentrate his chief efforts on keeping the patient alive, so that he may be relatively unconcerned with a fifth-nerve involvement.
3. Possibly the patient's bulbar poliomyelitis infection was so slight that it never proceeded to a clinical stage. (This is often referred to as the subclinical case.)
4. Someone may have failed to make the correct diagnosis, regardless of the severity of the disease.

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The subclinical and nonparalytic cases present a real problem. Howe, in a symposium on poliomyelitis,[8] states that for every recognized infection there are 100 unrecognized cases. Findings based upon the disease's peculiar local and world-wide distribution pattern reveal that natural immunity against poliomyelitis seems to diminish proportionately as living standards rise.[9, 10] The study of primitive populations, which have an insignificant record of clinical poliomyelitis,[11, 12] leads to some interesting speculations. These primitive peoples not only seem to possess a natural immunity against poliomyelitis, but early exposure during childhood seems to fortify them in addition with an acquired immunity.[9, 10] According to Jordan and associates[15] and Stevens,[16] it seems safe to assume that a great percentage of the adults in this country have had one or more bouts with poliomyelitis, since polio virus antibodies have been found in the sera of many test cases in which there was no history of polio infection.

The term "subclinical," particularly as applied to the unknown cases of poliomyelitis, has some serious shortcomings. The meaning seems clear—the symptoms of the disease have not reached the threshold of clinical recognition—but here lies the pitfall. Who should recognize the disease? (1) The patient, if he is old enough; (2) his parents or family, if they possess diagnostic abilities, intelligence, and interest; and (3) his physician, if he is consulted at all.

CASE REPORT

A 24-year-old white soldier was referred to me by the medical officer at an overseas Army dental clinic with the request that I help this man, who was unable to chew his food. The tall, somewhat underweight patient had a very striking appearance. His face looked extremely emaciated. The tissue covering his skull in the temporal region felt like parchment paper. His malar bones and zygomatic processes were very prominent and were contrasted by deeply sunken cheeks. His lower face was disproportionately long because of the position of his mandible, which was only "hanging" there. The patient could close his lips only with great effort. He appeared weak and tired but not in acute distress. His present complaint was that he was unable to close his mouth without using his hands and that the jaw would fall open as soon as he removed the auxiliary force. He demonstrated his technique of eating without his mandible; he manipulated food with his tongue against the upper teeth, which of course limited his diet. Extensive military maneuvers had been scheduled, and the patient was afraid that in his weakened condition he could not participate.

The previous history was most revealing. Eighteen months earlier, while still a civilian, the patient had developed a severe infection of a lower third molar. The tooth was extracted, but this did not relieve the infection which, instead, became worse. The patient experienced chills, ran a high fever, and vomited several times within the next few days. He received many penicillin injections, of unknown dosage, for his "dental" infection. A few days later, he recalled, his neck became sore and somewhat stiff; at the same time, he began to have difficulty in swallowing (not only food but also his saliva). His physician now decided on hospitalization. The patient was told that his difficulty in walking was caused by weakness, since he had not eaten properly for several days. In spite of intravenous feeding and various "shots" for his "dental" infection, the patient grew constantly weaker—so weak, in fact, that he lay in bed for weeks unable to move his head or his extremities. He did not recall having been in an oxygen tent or having lost consciousness during his confinement. Nor did he remember having had any swelling or

particular pain around the extraction site. After many weeks his strength slowly returned, and he could swallow again; only his face seemed weak from the dental infection, and he had difficulty in chewing. It took about a year for him to regain most of his strength.

Fifteen months after the fateful extraction, the patient was inducted into the Army, with an over-all profile of 1, indicating a perfect physical condition. After basic training, he was sent overseas. The patient related that his present trouble started with a mess sergeant who resented anyone's not eating the food which was served. The mess sergeant finally ordered this young man to the dispensary for medical evaluation.

Fig. 1.

Fig. 2.

Fig. 1.—Patient at the age of 22 years, shortly before his illness.

Fig. 2.—Patient, aged 24 years, at time of dental examination. Note temporal region, prominent zygomatic arch, sunken cheeks, elongation of lower face, and change in lip contour, reflecting the efforts of perioral muscles to keep the lips closed against the pull of the sagging mandible.

Digital examination verified the visual impression. Only parchmentlike tissue could be felt overlying the temporal bones. The cheeks were sunken and felt flaccid, with no discernible muscle tonus. The mandible hung open passively, leaving a distance of 18 mm. between the upper and lower incisors. The patient's speech was halting and required considerable effort. When asked to close his jaw, the patient first succeeded in elevating it a few millimeters with his perioral muscles. He had to use his hands for complete closure. There was a normal occlusal relationship when the jaws were held together. The intraoral examination was essentially negative; all teeth were present with the exception of the lower left third molar. The loss of his mother's special cooking, which had probably minimized the patient's chewing difficulties at home, finally forced attention to the serious problem.

With this type of history, one wonders how much is needed to promote a subclinical case to a clinical one. The fact of the patient's induction into the Army and his perfect Army medical record testifies to the extreme range of "subclinical" possibilities!

This case presented an almost classic picture of bulbar and spinal poliomyelitis. After extensive follow-up studies, the neuropsychiatric ward concurred with my tentative but late

clinical diagnosis, and the the complete bilateral atrophy of the masseter and temporalis muscles was confirmed electromyographically. Medical discharge was recommended for this patient, since no type of therapy was available to help him now.

DISCUSSION

Although dentists are not involved with the primary treatment of poliomyelitis, they may have to treat the residual effects where current therapeutic procedures have been unable to preserve sufficient muscle power. Aside from intraoral care, such patients as the one whose case is reported here will need either an elastic chin strap to substitute for the missing elevators or a chest-chin support. The latter is a plastic block which can be carried on a strap around the neck. It is formed in such a way as to allow the chin to rest firmly in it when the patient bends his head forward. It permits the patient to eat in a nodding fashion. It has the advantage of being less noticeable than the chin strap. In the foregoing case a combination of the two devices was recommended. In cases of unilateral paralysis, Mühlemann[3] recommends that interdental appliances be worn at night to prevent permanent contracture of the normal muscle. Surgical transplantation of tendons, which has been successful in cases involving the extremities, has not yet been perfected for the muscles of mastication.[13, 14]

SUMMARY

The dentomedical implications of residual effects of bulbar poliomyelitis have been discussed. A case of complete bilateral atrophy of masseter and temporalis muscles reflecting the tragic dental effects of the disease has been reported.

Hope has been expressed for the possible prevention of residual damage by early diagnosis and treatment of polio patients. This will require greater cooperation among the healing professions as well as help from a better-educated public.

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A TUBERCULOUS GRANULOMA OF THE ORAL MUCOSA AND CERVICAL LYMPH NODES

Report of a Case

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TUBERCULOSIS belongs to a group of diseases referred to as infectious granulomas, the term "granulomas" referring to the granulation-like appearance of the tissue reaction which characterizes these infections. The group includes syphilis, tuberculosis, sarcoidosis, leprosy, actinomycosis, and other mycotic and funguslike infections. Although the literature describes many definite and specific microscopic characteristics for each of these diseases, their clinical variations are many and are multiplied even more by the presence of secondary infection.

The lesion of tuberculosis is the result of an interplay of the forces of destruction and repair, plus the additional factors of the host's sensitivity to the organism and the virulence of the organism itself. The tendency to repair, however, is commonly greater than the destructive process. The lesion can be seen in several stages of development. Polymorphonuclear leukocytes are the first cells to arrive in response to the tubercle bacilli. They are actually phagocytic but are unable to damage the organism they engulf. Within twenty-four hours the polymorphs are replaced by macrophages and monocytes. This is the body's chief response to the bacillus; however, it is a response to the organism's lipoid coating rather than to the bacillus itself. The monocytes engulf the polymorphonuclear leukocytes and their contained organisms, and a gradual dispersion of the lipoid material takes place within the cytoplasm of the monocytes, transforming them into so-called epithelioid cells. Giant cells are formed by the fusion of a number of epithelioid cells, but only in the presence of necrosis. They attain a large size and contain peripherally arranged nuclei or clumps of nuclei at one or both poles.[1]

Involvement of the mucosa of the oral cavity or the cervical lymph nodes is usually found in patients suffering from active pulmonary tuberculosis. The organisms are carried in droplets of sputum into the oral cavity from the tracheobronchial tree. There have been reported, however, cases of localized tuberculous

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cervical adenitis[2] without pulmonary disease. There is much speculation as to the possible portal of entry in such cases.

The following is a case report of a localized tuberculous granuloma of the oral mucosa and cervical lymph nodes without evidence of pulmonary disease. The additional factors of long-standing secondary infection and arteriovascular lues further complicated the differential diagnosis in this case.

CASE REPORT

History of Present Illness.—A 39-year-old Negro woman was seen in the oral surgery clinic of Western Pennsylvania Hospital on Jan. 7, 1959, with the chief complaint of a "painfully ulcerated mouth and swollen jaw." Two years earlier, following extraction of all her remaining teeth, she noticed an enlargement of the submental nodes. These nodes had remained enlarged to this date. She stated that three months prior to her appearance at this clinic, a penny-sized ulcer developed in the left mandibular buccal fold. This had slowly progressed to its present size. She also stated that six months previously she began experiencing fainting spells and that they had seemed to occur more frequently since the onset of her oral complaint. She had not experienced any of these so-called "spells" for the last three days.

Fig. 1.—Ulcerated, granular lesion running along the buccal surface of the mandibular alveolar ridge.

Clinical examination revealed an ulcerated, granulating, nonfungating lesion in the left mandibular buccal fold. It involved an area extending from the third molar region anteriorly to the left lateral incisor. There was considerable evidence of secondary infection; its borders were slightly indurated, and it appeared attached to the underlying bone at several points (Fig. 1). The patient was completely edentulous and had never worn a full denture prosthesis. The tonsillar area and oropharynx showed no evidence of ulceration but were slightly injected. The tongue and the remaining oral mucosa were within normal limits. The patient did not complain of trismus or dysphagia. There was

no loss of sensation along the distribution of the inferior alveolar nerve or its mental branches, but the patient did exhibit a slight degree of motor weakness in the area supplied by the mandibular branch of the facial nerve. The submental nodes were markedly enlarged, nontender, and of a rubbery consistency. There was also a similarly enlarged node that could be palpated in the left submaxillary region. The associated soft tissue of the area was swollen and tender (Fig. 2).

Lateral oblique and anteroposterior films of the mandible, as well as intraoral films, were ordered at this time. Wet readings gave no indication of bony involvement. With 2 c.c. of Xylocaine hydrochloride, a block of the left inferior alveolar nerve was obtained. An additional 0.5 c.c. was infiltrated in the vicinity of the long buccal nerve. A wedge of tissue was excised with a No. 15 blade; this included part of the lesion, its indurated border, and adjacent normal-appearing tissue. The wound was closed with three 0000 black silk sutures. The specimen was submitted in 10 per cent formalin for histologic study. The patient was then admitted to the oral surgery service of this hospital.

Fig. 2.—Clinical enlargement of the submental and submaxillary nodes.

Medical History.—Because of some contradictions in the patient's story and a lack of obtainable past medical records, there was some difficulty in obtaining a reliable history. The patient did remember several episodes of urinary tract infection two years earlier, "shots" she received about one year before for "spasms," and the fact that she had received treatment for hypertension from a local physician and was on a salt-free diet. She revealed no history of productive cough, dyspnea, angina, orthopnea, night sweats, increased thirst, frequency of urination, or swelling of the ankles. She did complain of a steady weight loss, frequent daily headaches, dizziness, and difficulty with vision; she said that her eyes ran constantly. There was no history of allergy, asthma, or hay fever. She had never experienced any episodes of severe prolonged bleeding. She denied any antiluetic therapy or a history of positive serology.

Physical Examination.—Physical examination revealed a fairly obese, confused, Negro woman who walked with a shuffling gait. Examination of her eyes revealed a slight ptosis of the left lid, with an associated conjunctivitis. There was a small, flattened, tannish nodule

at the junction of the sclera and the iris. Other neurologic findings included indistinct optic disc margins and a slight left central facial nerve weakness. Other findings included a blood pressure of 130/95; pulse, 88; respirations, 20; and oral temperature, 99.2° F. The heart was slightly enlarged but within normal limits. The lungs and chest were clear to percussion and auscultation. No palpable abdominal masses were found. There were no other significant physical findings.

Laboratory Findings and Diagnostic Procedures.—The results of tests ordered on admission were as follows: hemoglobin, 12.2 Gm.; hematocrit, 39 per cent; white blood cells, 7,000. The differential count was neutrophils 62 per cent, lymphocytes, 30 per cent, monocytes 3 per cent, eosinophils 1 per cent, and 4 band cells were seen. Bleeding time, coagulation time, serum electrolytes, nonprotein nitrogen, blood sugar, alkaline phosphatase, and urinalysis were all within normal limits. A dark-field smear taken from the oral lesion failed to demonstrate the presence of *Treponema pallidum*. Acid-fast examination of the sputum was negative. VDRL studies exhibited a 4 plus reaction and a positive Kolmer test. The serum electrophoresis revealed a marked increase in beta and gamma globulins. The report stated that this response could be due to active syphilis, sarcoidosis, or liver disease, but probably not to active tuberculosis. The electroencephalogram revealed no significant abnormalities. Films of the skull, chest, and bones of the hands and feet likewise revealed no significant abnormalities.

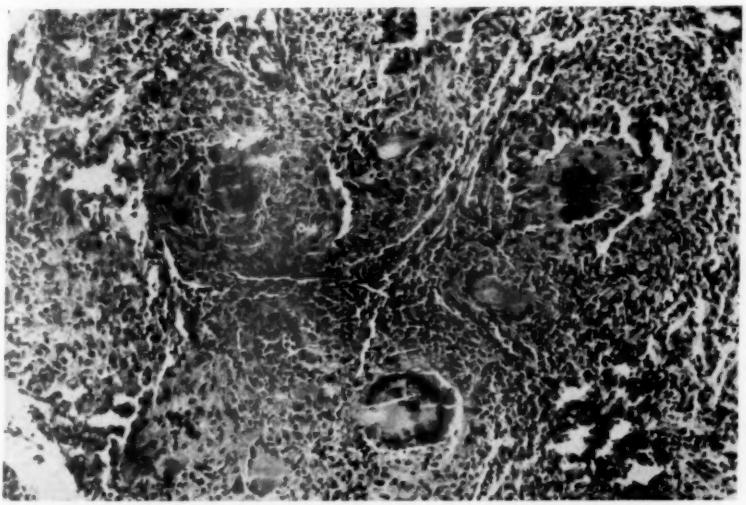


Fig. 3.—Active tuberculous granuloma of oral mucosa. Note foreign-body giant cells among clusters of epithelioid cells. (Magnification, ×150; reduced ⅓.)

A biopsy report of the tissue sent from the pathology department described a specimen submitted in formalin consisting of a wedge-shaped irregular granular fragment of tissue measuring 0.8 cm. in its greatest diameter. The microscopic report described clusters of epithelioid cells that were surrounded by a chronic inflammatory type of exudate. There was no evidence of caseating necrosis, but there were numerous giant cells of the foreign-body or Langhans type in the clusters of epithelioid cells. Sections submitted for periodic acid-Schiff and acid-fast stains were negative. The diagnosis indicated by these findings was active granuloma of the oral mucosa (Fig. 3).

Clinical Course.—On January 11 the patient was scheduled for a rebiopsy of the oral lesion. However, she experienced a grand mal type of seizure which lasted for two or three

minutes. She was placed on phenobarbital and Dilantin therapy, and convulsion precautions were taken. It was thought wiser to delay the biopsy procedure until the patient's present condition had become more stabilized. The patient was placed on a combined oral surgery, neurosurgical, and medical service.

On January 12 the patient experienced two abortive seizures, at which time additional phenobarbital and Dilantin were administered.

On January 14 the patient was given a liquid breakfast but was allowed to have nothing by mouth after 9 A.M. At noon she was given 50 mg. of Phenergan hydrochloride intramuscularly and a soapsuds enema. At 1 P.M. she received hypodermic injections of 75 mg. of Demerol hydrochloride and 1/100 gr. of atropine sulfate. She was placed on call to the operating room for 2 P.M.

Under Surital sodium and nitrous oxide and oxygen anesthesia, with endotracheal anesthesia administered via an oral route, the patient was prepared and draped in an appropriate manner. The oropharynx was packed, and two wedges of tissue were removed. One specimen was sent for a repeat histologic study and periodic acid-Schiff and acid-fast stains. The other section was sent for culture, smear, and guinea pig inoculation.

The patient was then turned carefully onto her left side, redraped, and prepared for a spinal tap. The fluid pressure measured 380 mm. of cerebrospinal fluid. A sample was sent for dark-field examination, serology, protein, and colloidal gold cure. The patient was then placed again in a supine position and carefully evaluated. She was very gently removed from the operating table and sent to the recovery room, where extubation was eventually done by the anesthesiologist. The patient tolerated the procedure very well. She was placed on the usual post-spinal tap precautions.

On January 15 the patient was placed on antiluetic therapy of 3,000,000 units of penicillin daily for ten days.

On January 19 spinal fluid serology and acid-fast studies were found to be negative, and all other studies were within normal limits. An angiogram revealed a slight shift of the anterior cerebral vessels to the left. The high spinal fluid pressure made it inadvisable to attempt a pneumoencephalogram. The neurosurgical note suggested the possibility of an expanding lesion in the right frontal lobe area.

On January 21 a purified protein derivative gave a negative tuberculin reaction.

On January 23 a second purified protein derivative gave a positive tuberculin reaction which produced a quarter-sized pruritic wheal.

On January 24 a ventriculogram revealed no significant abnormalities. The patient had not experienced any seizures since January 12.

Operative Procedure.—On January 27 the patient was taken to the operating room for excision of the submental node. The submaxillary node palpated on admission had regressed under the antibiotic therapy, as had the associated secondary infection. The submental node failed to respond to treatment.

The patient was prepared and draped in an appropriate manner. After anesthesia had been obtained by the infiltration of 1 per cent procaine hydrochloride with 1:100,000 epinephrine, a transverse incision was made over the node. Several nodes were then selected and isolated by blunt dissection and freed. Hemostasis was secured with multiple ties, and the wound was closed with interrupted 00000 chromic catgut sutures subcuticularly, followed by 000000 interrupted black silk sutures to close the skin. A small pressure dressing was applied and the patient was returned to her ward.

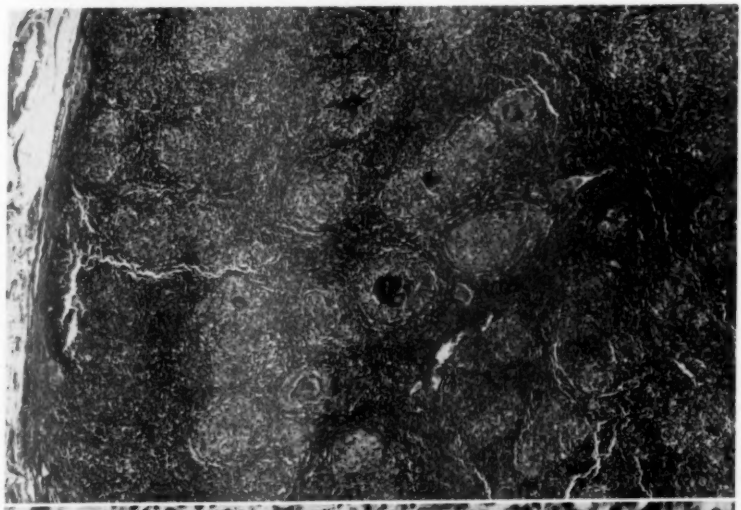
The specimen was submitted in two parts—one placed in 10 per cent formalin for histologic study and periodic acid-Schiff and acid-fast stains and the other sent for culture and guinea pig inoculation.

Summary of Pathology and Bacteriologic Studies.—Both the initial biopsy on admission and the repeat biopsy of the mucosal lesion were found negative for fungi and acid-fast organisms. The only positive bacteriologic findings were the presence of penicillin-sensitive *Streptococcus viridans* and hemolytic *Staphylococcus albus.* The histologic pictures were

identical, both indicating an active granulomatous type of infection. The immediate smears did not reveal any fungi, spirochetes, or acid-fast organisms, and further culture and guinea pig inoculation were instituted.

The pathology report on the submental nodes removed on January 27 was as follows: "The specimen consists of two closely adherent, discrete, hemorrhagic, thinly encapsulated lymph nodes. Microscopic examination reveals discrete and confluent nodules of epithelioid cells which have obliterated the normal architecture of the node. These areas lack caseation

Fig. 4.



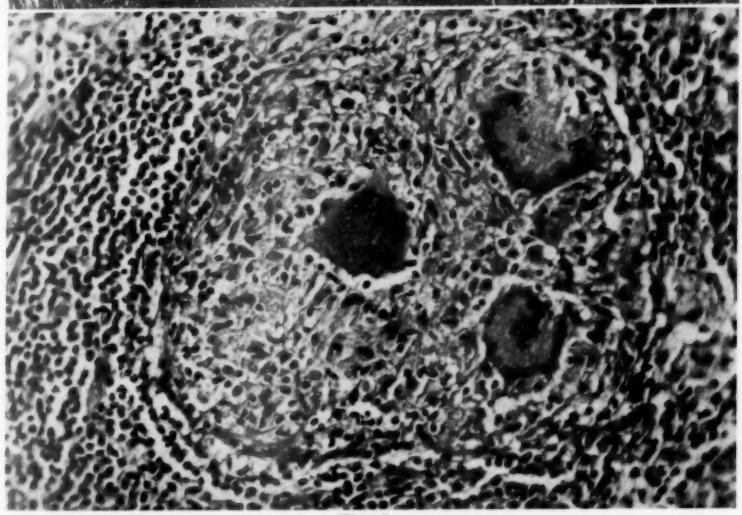


Fig. 5.

Fig. 4.—Microscopic section from submental node shows lighter epithelioid nodules infiltrating the normal architecture of the node. (Magnification, ×50; reduced ⅓.)
Fig. 5.—High-power photomicrograph of epithelioid clusters and giant cells (Langhans' cell) with periphery of lymphocytes. (Magnification, ×300; reduced ⅓.)

and are surrounded by a zone of chronic inflammatory cells. There are many foreign-body giant cells scattered within the epithelioid zones. The microscopic diagnosis is compatible with sarcoidosis." The periodic acid-Schiff and acid-fast stains were negative (Figs. 4 and 5).

On January 30 the patient was free of her seizures and all swelling had subsided; the oral lesion, although the same size, was now free of secondary infection. The patient was then discharged, to be followed on an outpatient basis.

Outpatient Follow-up.—On February 5 every other suture was removed. The wound appeared to be healing well. All remaining sutures were removed on February 9.

On February 20 the culture started from material taken from the submental node revealed the presence of tubercle bacilli, and a small nodule could be palpated in the guinea pig inoculated with material from the oral lesion.

Fig. 6.—Marked regression of ulcerated area following antituberculin therapy.

On February 26 the guinea pig was killed, and the node in question was isolated and studied microscopically. Tubercle bacilli were demonstrated in this specimen, and antituberculosis therapy was instituted.

On March 10 there was marked regression and resolution of the mucosal lesion (Fig. 6).

DISCUSSION

With the positive culture, repeat chest films were ordered and antituberculin therapy was instituted. The chest films were again negative. There had been no recent onset of chronic cough or bloody sputum, and the chest was still clear to auscultation and percussion. The diagnosis made at this time was tuberculous granuloma of the oral mucosa and submental lymph nodes.

From the time of the first biopsy report, all who were concerned with this case realized that we were dealing with one of the granulomatous infections, that is, mycotic infections, lues, tuberculosis, or sarcoidosis. The mycotic infections were eliminated by repeated cultures and periodic acid-Schiff stains. The

serum electrophoretic pattern accentuated the possibility of a luetic process or sarcoidosis and, in fact, masked our thinking in the statement that the rise in beta and gamma globulins was not likely to have been produced by a tuberculoma. The neurologic symptoms could be attributed to lues, sarcoidosis, and tuberculosis. In fact, the literature contains excellent examples in which these symptoms of epileptiform seizures, headache, eye signs, facial weakness, and dragging gait were found in studies made on each of these diseases. All were reported to have mucosal and node incidences. The initial lack of acid-fast organisms is not a remarkable finding. Frequently the organism cannot be isolated by direct-smear techniques. Prolonged culture on special media and guinea pig inoculation are needed to concentrate the bacillus in sufficient numbers for it to be demonstrated. The microscopic sections revealed a fairly typical sarcoid lesion. However, tuberculomas are quite similar to this picture—so similar that there are investigators who believe that the lesion of sarcoid is caused by an attenuated tubercle bacillus. Others believe that 20 per cent of the patients with sarcoid have concurrent tuberculosis. The most confusing element in this case may be attributed to the coincidental presence of two granulomatous infections, lues and tuberculosis. Syphilis has long been known as an imitator of many disease processes. The suspected brain lesion is most probably a manifestation of a now arrested and latent tertiary stage of syphilis.

SUMMARY

A case of tuberculous granuloma of the oral mucosa and submental nodes, with additional findings of latent cerebrospinal lues, has been presented.

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HERPES ZOSTER OF THE MANDIBULAR NERVE

Report of a Case

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HERPES ZOSTER, sometimes referred to as shingles, zoster, zona, ignis sacer, and hemizona, may occur as a primary acute infectious disease or as a secondary disease process caused by luetic or tubercular lesions.[2] Tumors or other neural lesions may also be causative factors in the secondary type.

The primary form is characterized by inflammation of one or more dorsal root ganglia or extramedullary cranial nerve ganglia.[1] Present evidence strongly suggests that the etiology is viral in nature, although experimentally the causative agent has not been definitely demonstrated. The most direct indications of this viral factor are the presence of intranuclear inclusions in the cells of the surface lesion, reported success in experimental transmission and the presence of immunity following the disease. It is associated with a prodromal period (usually of three or four days' duration) of malaise, elevated temperature, and pain. This is followed by a vesicular type of eruption which appears along the peripheral distribution of the affected nerve. Regional lymphadenopathy may be present. The febrile period lasts from three to five days. Finally, subsequent drying and healing of the cutaneous lesions begin. Secondary infection may delay healing and increase the tendency to scarring. Neuralgic pains may persist, especially in elderly persons, and there may be some residual paralysis at times.

Histopathologically, the lesion consists of severe inflammation and destruction of the ganglion cells and fibers.[2] The cutaneous vesicle is situated in the epithelial layer, and there is marked inflammation of the corium. Acidophilic intranuclear inclusions are found in the epithelial cells around the vesicle. The microscopic picture of chickenpox and herpes zoster is indistinguishable, adding to the clinical and epidemiologic evidence of the close relationship between these two diseases. The virus is distinct, however, from the organism that causes herpes simplex.

Herpes zoster involving the trigeminal ganglion most commonly involves the ophthalmic branch, while the mandibular branch is the least often afflicted. The postherpetic neuralgia may assume many of the characteristics of tic

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douloureaux. Here the history of skin lesions, visible cutaneous scars, and possibly some sensory impairment aid in the differential diagnosis.

CASE REPORT

History of Present Illness.—An 85-year-old white woman was seen in the emergency room of The Western Pennsylvania Hospital on Aug. 10, 1958, with the chief complaint of a painfully infected mouth. She stated that she was experiencing a sharp, radiating pain along the left side of her face and in her left ear. She explained further that for the last three or four days she had not been feeling well and had been running a slight fever. About two days prior to her appearance at this institution she had felt a severe burning, tingling sensation that began in the vicinity of the left mental foramen and traveled along the alveolar ridge over the lip and out over the entire left mandibular body and ramus. Her lip began to swell on the afflicted side and multiple tiny ulcerations appeared along the buccal mucosa. Subsequently, lesions appeared on the external surface of the lip and seemed to spread to the skin overlying the left mandible. The patient had been unable to take any nourishment since the onset of her severe pain.

Fig. 1.—Herpetic vesicles in area of lip, symphysis, and mental foramen.

Clinical examination at this time revealed a vesicular type of eruption which began at the midline of the lower lip and symphysis and extended along the skin of the face overlying the body and ramus of the left side of the mandible. Some vesicles could be seen on the skin of the external ear. In places the vesicles were beginning to become encrusted (Figs. 1 and 2). On palpation, the submaxillary and cervical lymph nodes were found to be enlarged and tender on the afflicted side. Oral examination revealed a completely edentulous mouth. The patient stated that she had been wearing her present dentures for at least fifteen years. The mandibular alveolar ridge was quite resorbed, but this was not remarkable in view of the patient's age and her long use of a full denture prosthesis. The buccal mucosa and the buccal alveolar ridge mucosa on the left side were covered with multiple yellowish ulcerations that were rimmed with an erythematous margin (Fig. 3). The lingual mucosa and tongue were not involved, and the rest of the oral cavity was not remarkable.

A tentative diagnosis of herpes zoster of the mandibular nerve was made at this time.

Past Medical History.—The patient stated that, except for recurrent episodes of gastrointestinal disturbances, she had never been seriously ill. She denied any history of exertional dyspnea, orthopnea, chronic productive cough, or chest pain. She knew of no drug or food idiosyncrasies or sensitivities. She did admit to an extremely poor dietary regime and to periods of extreme nervousness. A review of old charts that were obtained from the outpatient department of this institution revealed that an electrocardiogram taken in November, 1954, and a chest film were found to be within normal limits and that a gastrointestinal series in 1955 confirmed a diagnosis of diverticulosis of the sigmoid colon.

Fig. 2.—Vesicle distribution along the ramus and on the skin of the external ear.

Physical Examination.—The patient was a frail elderly white woman in acute distress. Her skin was dry and warm. She had a temperature of 100.5° F. There was no evidence of ecchymosis or dependent edema. The pulse rate was 100 per minute; blood pressure was 136 systolic and 75 diastolic; and respirations were recorded as 23 per minute. The chest was clear to auscultation and percussion.

Laboratory Findings.—Blood studies revealed the following: hemoglobin, 10.4 grams; hematocrit, 32 per cent; white blood cells, 4,500. The differential count was neutrophils 68 per cent, lymphocytes 31 per cent, and monocytes 1 per cent. Urinalysis disclosed a specific gravity of 1.012, a pH of 6.2, a 1 plus acetone reaction, and negative sugar and albumin reactions.

Lateral-jaw films and posteroanterior views of the mandible, as well as intra-oral and occlusal views, failed to show anything significant.

Treatment and Clinical Course.—The patient was placed on a supportive and therapeutic regime. Phenergan, 50 mg., and Demerol, 25 mg., were administered immediately for the relief of pain. It was felt that heavy narcosis would be undesirable in view of the patient's age. She was given Percordan tablets to be taken every four hours if needed for relief of pain, and Compazine, 10 mg. three times daily, was ordered. To prevent secondary infection, 250 mg. of Mysteclin every six hours was prescribed. The patient returned daily for the administration of one ampule of Protamide. She was instructed to force fluid intake and to take one Becotin capsule with each meal.

Consultation requests were submitted to the medical, dermatology, and ear, nose, and throat services. The ear, nose, and throat service reported that otoscopic examination re-

veiled minute ulcerations on the anterior surface of the tympanic membrane, and 2 per cent hydrogen peroxide solution to be used as an ear irrigation three times a day was prescribed. The medical and dermatology services agreed with the diagnosis of herpes zoster and with the present course of therapy.

On October 14 a repeat urinalysis revealed negative reactions for sugar, acetone, and albumin. A temperature reading of 98.8° F. was obtained. The patient complained of recurrent episodes of pain; however, they were of decreased duration and severity. The oral lesions were beginning to fade, and the cutaneous lesions were all encrusted. No new crops of vesicles had appeared.

Fig. 3.—Mucosal lesions on inner surface of the lower lip and along the buccal mandibular vestibule.

On October 19 the patient was found to be afebrile, and all lesions were beginning to fade. Otoscopic examination revealed complete clearing of the tympanic membrane. Antibiotic therapy was cut, as well as the Protamide injections. The Becotin dose was cut to two capsules a day. There was improvement in the patient's general outlook, but she still complained of some residual pain at the inferior border of the mandible and in her left ear.

On October 25 the patient was seen in the oral surgery clinic. All lesions had disappeared by this time. The patient stated that she was quite comfortable now but that she did experience some mild pain in her left mandible from time to time. An appointment was made for follow-up photographs. The patient failed to keep her appointment, however, and she has not answered an inquiry sent to her by mail.

CONCLUSION

A case of herpes zoster with involvement of the mandibular nerve has been presented. As with many of the viral infections, treatment is at best palliative. The use of Protamide, a denatured enzyme of animal gastric mucosa, finds its rationale in shortening the acute phase and lessening the severity of the inflammation. The pain is quite severe and disabling, and in elderly patients it may persist long after the cutaneous and mucosal lesions have vanished.

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PHARMACOLOGY AND THERAPEUTICS

THE ROLE OF SALIVARY DEPRESSANTS IN DENTISTRY

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THE presence of copious quantities of saliva in the oral cavity during dental operations is a major problem. Investigations have shown that undesirable chemical and physical changes occur in dental materials contaminated by saliva. The rubber-dam and other aids have not been universally employed to prevent contamination. Even when the most modern aspirating devices are used, cementation of bands or crowns, insertion of restorations, and other dental procedures are hampered by the presence of excessive saliva. The visibility of the operative field is also limited.

For many years the medical profession has used drugs to control excessive secretions.[1, 2] Hospitalized surgical patients are given hypnotics, sedatives, or analgesics prior to the administration of anesthesia. Atropine, scopolamine, Demerol hydrochloride, and other similar medications are used. It is recognized that the side effects of these drugs are not desirable for the ambulatory dental patient. For the average dental patient, any agent that reduces the flow of saliva should meet certain general requirements; it should be safe, easy to administer, quick-acting, of limited duration, specific, and compatible with other drugs routinely administered in the dental office, and it should produce a minimum of side effects.

LITERATURE

Research investigators in anesthesiology[3-6] have reported numerous chemical means of controlling saliva, and several have indicated that these medications offer potential advantages in dental operations.

Weise,[7] in 1927, suggested the use of methylatropine prepared for submucous injection. He reported that ten minutes after the injection of 0.1 to 1 mg. of "Salivan" there was an inhibition of salivary flow which lasted for one to two hours. The author realized it would be helpful to control excessive saliva for dental procedures. In 1951 DeBre[8] reported on the use of Banthine in 100 dental patients. He stated that ninety-six patients demonstrated beneficial effects from a 50 mg. dose; dryness of the mouth occurred in four to

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fifteen minutes and lasted from one to three hours. No side effects were observed from a single administration. In 1953 McDonald[9, 10] reported the effects of antihistamines on the flow of saliva. Although his study was not directed toward dental application, it indicated that Chlor-Trimeton reduced salivary flow 25 per cent and that Benadryl and Pyribenzamine depressed the flow about 20 per cent. Masters,[11] in 1959, tested the levo-hyoscyamine drug, Bellafoline, clinically to determine its effectiveness as a dental antisialogogue. Bellafoline, which contains all the levorotatory alkaloids of belladonna, had fewer side effects than other atropine-like drugs; it was administered according to the degree of salivation as determined by visual observation. Newton and Morrison[12] reported that methantheline bromide produced the greatest depression of saliva and the most rapid onset, whereas scopolamine hydrobromide and atropine sulfate were less effective.

PROCEDURE

In this study four drugs were selected for clinical evaluation and a placebo was utilized as the control. The drugs in their manufactured form were given to the Pharmacology Department of Tufts University School of Medicine for coding and packaging. All drugs, including the placebo, were packaged in similar containers to standardize size, color, and general appearance and were coded 0 to IV. The code and dosage were as follows:

0. Promethazine hydrochloride (Phenergan), 25 mg.
I. Methantheline bromide (Banthine), 50 mg.
II. Tricyclamol chloride (Elorine), 50 mg.
III. Placebo, 300 mg. capsule of lactose
IV. Meprobamate (Equanil), 200 mg., plus methantheline bromide (Banthine), 50 mg.

A colored 300 mg. capsule was selected for use and lactose, an inert material, was added to provide uniform appearance and masking. Each drug capsule was put into a small coin envelope which, in turn, was placed within a larger one. The patient's number was written on the larger envelope, while the visit number as well as the patient's number was noted on the smaller one. The investigator was not aware of the significance of the code until the study was completed and the data were analyzed.

The subjects, 8 to 12 years of age, were selected from the Oral Pediatrics Department of Tufts University School of Dental Medicine and randomized by use of a Latin square table of five. Twenty-two volunteers were selected with the stipulation that each patient must be present for five hours, the time required for an evaluation. During the visit, the patient was isolated with reading material or school work to occupy his attention and to limit his activity. Sugar-free gum was given to the patient as a salivary stimulant.[13-15] An initial examination was conducted by the physician before and one hour after administration of the medication. The patient was available for a period of two hours for the collection of saliva samples by the "modified Mushin" technique[16]

in combination with the Curby apparatus.[17] A minimum of three test readings were taken prior to administration of the medication.

During the collection of saliva samples the patient was first asked to swallow and then requested to expectorate repeatedly into a graduated cylinder for a one-minute period. A drop of caprylic alcohol was added to the tube for the purpose of dispersing foam. The volume was read to the nearest 0.5 ml., and each measurement was recorded at five-minute intervals to determine as closely as possible the onset of action. If the time interval exceeds two hours, any drug of this nature is of limited value to the dental profession; therefore, the readings were continued for only two hours from the initial administration of the medication. Subsequently, the patient had routine dental procedures performed at the clinic. He then returned for additional saliva samplings. By this means, it was possible to evaluate the time of onset, the duration, and the effectiveness of the drugs.

The senior resident of the Boston Floating Hospital examined each patient in order to evaluate his physical status, to determine the effectiveness of the drug, and to note any side reactions. The routine physical examination made at each visit consisted of pulse rate, blood pressure determinations, and throat and eye examinations. The eye examination was recorded in detail and the pupil was evaluated for size, shape, reaction to light, and accommodation.

Prior to administration of the drug each patient was given two glasses of water in order to reduce the possibility of urinary retention. The parents were asked to note any problems of this nature following dismissal of the patient.

The data were then given to the statistician for analysis.

RESULTS

This study was conducted for the purpose of evaluating a series of drugs as salivary depressants. Four drugs plus the placebo were administered to a total of twenty-two patients, and the volume (ml./min.) at greatest depression, onset of greatest depression, and duration of salivary depression were measured and recorded as shown in Table I.

TABLE I. DENTAL EVALUATION

NUMBER OF PATIENTS	CODE AND DOSAGE	ONSET OF GREATEST DEPRESSION (MINUTES)	DURATION (MINUTES)	VOLUME (ML./MIN.) AT GREATEST DEPRESSION
22	0. Promethazine hydrochloride (Phenergan), 25 mg.	72	30	1.8
22	I. Methantheline bromide (Banthine), 50 mg.	60	120+	0.52
22	II. Tricyclamol chloride (Elorine), 50 mg.	80	60	1.25
22	III. Placebo, 300 mg. capsule of lactose	105	No change	1.48
22	IV. Meprobamate (Equanil), 200 mg., plus Banthine, 50 mg.	90	120+	0.55

The total quantity of saliva from the control patients was 1.48 ml./min., as compared to reduced amounts from the medicated groups. Banthine reduced the salivary flow to 0.52 ml./min., the most significant reduction achieved with any of the drugs tested.

The results demonstrated that the period of onset of the greatest depression extended from 60 to 90 minutes, with Banthine showing the most rapid action and Banthine plus Equanil displaying the slowest onset. The duration was longest when Banthine or Banthine plus Equanil was administered; the results indicated that these drugs depressed salivary flow for at least two hours. Elorine depressed the flow of saliva for approximately one hour, while Phenergan produced a reduction for only thirty minutes after onset (Fig. 1).

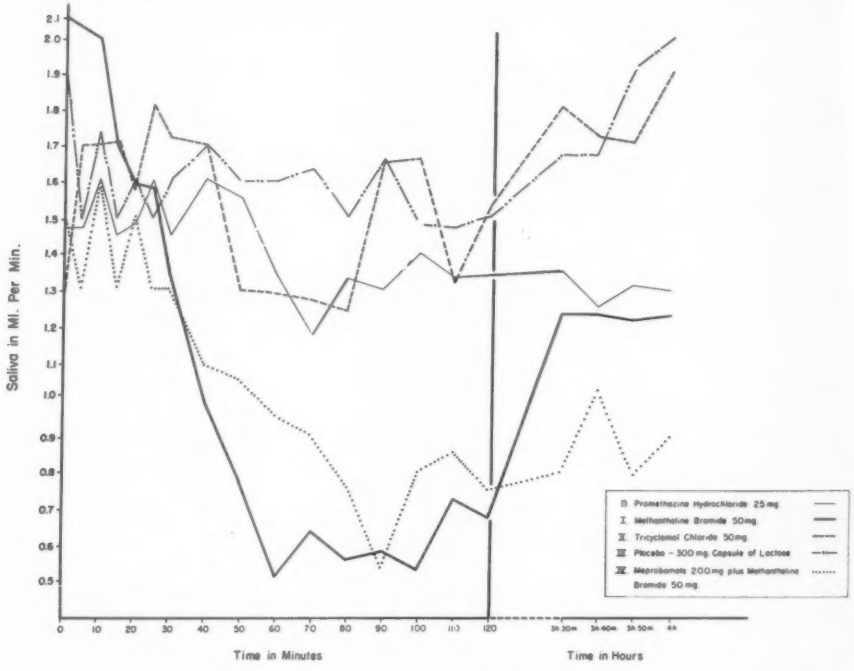

Fig. 1.—Saliva flow rate after drug administration.

The medical examinations (Table II) performed previous to and one hour after administration of the drug took about one-half hour at each visit. It can be seen from the table that the pulse rate decreased in the majority of patients tested. With the administration of Banthine, however, nine patients demonstrated an increase in pulse rate, eight showed a decrease, and three had no change. Diastolic and systolic pressures were measured and recorded. A significant difference was found at the 5 per cent level between the placebo and the test drugs. A probable psychosomatic effect from administration of the placebo

demonstrated that the distribution of change in blood pressure was equal in the three areas showing increase, decrease, and no change. It was noted that when Banthine was used there was a systolic increase and a diastolic decrease.

The visual acuity and pupil size were measured before as well as one hour after administration of the drug. The results generally showed no significant changes, but Banthine produced a slight increase in visual acuity and in size of the pupil.

The physician was requested to observe and record the state of dehydration of the oral cavity. Banthine alone was found to produce a comparatively dry condition.

TABLE II. MEDICAL EVALUATION

	PULSE			BLOOD PRESSURE						VISUAL ACUITY			PUPIL SIZE			HYDRATION		
				DIASTOLIC			SYSTOLIC											
DRUG	+	−	N.C.	+	−	N.C.	+	−	N.C.	+	−	N.C.	+	−	N.C.	+	−	N.C.
0	2	18	0	2	6	12	5	3	12	5	4	12	1	3	16	0	7	15
I	9	8	3	1	7	13	8	2	11	8	7	13	6	1	14	0	13	8
II	1	18	1	2	6	12	5	5	9	1	2	14	1	3	18	0	5	17
III	5	15	2	6	8	8	7	6	9	3	2	16	2	0	20	0	3	19
IV	5	14	2	2	5	14	2	3	16	3	2	16	5	1	14	0	8	14

+ = Increase.
− = Decrease.
N.C. = No change.

The parents supplied information concerning dryness of the mouth and urinary retention. They reported that the patients requested more drinking water than usual but did not complain of the existing dryness. According to the parents, there was no urinary retention.

It is possible that these drugs produced side reactions at the dosage used, but the investigators were unable to detect them by the methods employed.

DISCUSSION

This study has shown that the salivary depressants reduce the flow sufficiently to warrant their use in dentistry where a dry field is helpful. It has also demonstrated that clinical observations of the action of drugs, the standardized double-blind technique, and cooperation with medical colleagues are invaluable in the testing of new drugs for use in dentistry. It is obvious that this work is best performed in institutions where carefully controlled studies and evaluations can be made. In addition, other drugs, different dosages, and a greater range in the ages and numbers of patients should be considered.

The apparatus used to measure salivary flow is extremely accurate and eliminates many variables. When the medication becomes effective, however, it is sometimes difficult to maintain the cup in position. This problem can be controlled by using a solution of glycerin and water around the outer ring to provide greater suction. The "modified Mushin" technique is an acceptable method for measuring flow rate after drug administration.

The medication, administered in capsule form, was satisfactory, although some of the younger patients had difficulty in swallowing it. This problem

might be alleviated by administering the drug in suspension or tablet form. In this study, the capsule was used exclusively.

All patients were isolated when the tests were made. This was done to prevent competition between patients and to eliminate any distraction which might influence the accuracy of the data.

According to the results, Banthine had the most rapid onset (sixty minutes), it reduced salivary flow to 0.52 ml./min., and its action endured for more than two hours. The onset of the other depressants ranged from sixty to ninety minutes. Phenergan was effective for thirty minutes, and Elorine for sixty minutes. The depressant action of these two drugs on salivary flow was not as great as that demonstrated by Banthine. In spite of the delayed onset of action of Phenergan and Elorine, it is possible that the degree of depression and the reduced length of action may be advantageous for some dental procedures. Banthine, however, with its rapid onset, long duration, and reduced volume of saliva, appeared to be the most effective. This corroborates the findings of Newton and Morrison.

It is traditional and desirable that patients receiving dental treatment become ambulatory following the administration of medication. Although, as stated previously, no harmful side reactions were noted following the administration of these salivary depressants, the dentist must be aware of these potential effects.* Disorientation and hallucinations have been reported in the literature following the use of scopolamine, atropine, and other related drugs.[18] Therefore, it is important that, before any drug is administered in the dental office, a complete understanding of its effects be known.

SUMMARY

1. Salivary depressants are useful in dentistry when a dry field is required.

2. Five different medications, including a placebo, were tested on twenty-two subjects, aged 8 to 12 years, of approximately the same body weight.

3. The method of evaluation and the apparatus employed were satisfactory for this study.

4. The medical examination determined the physical reactions to the administration of these salivary depressants. A reflex reaction test should be added to the physical examination to determine the presence of drowsiness.

5. Banthine was the most effective salivary depressant, but Phenergan and Elorine may be advantageous for some dental procedures.

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*It would be helpful to add a reflex reaction test to the medical examination in order to determine the presence of drowsiness. It was observed that Phenergan produced this reaction in a few patients.

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ORAL ROENTGENOLOGY

American Academy of Oral Roentgenology

Arthur H. Wuehrmann, Editor

CONSIDERATION OF LONG-RANGE OBJECTIVES FOR THE TEACHING OF UNDERGRADUATE RADIOLOGY IN DENTISTRY

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DESIGNING a new curriculum involves essentially the same disciplines that are utilized in planning a research project. In reality, a proposed curriculum is a research program in education. It should be emphasized that the realistic approach to new curriculum construction infers that such a program can be implemented in the near future. This philosophy reflects a concept of future growth in the light of present needs.

Inasmuch as radiology functions in various degrees as a common denominator in the teaching activities of all departments in the dental school, these broad needs must be considered in the organization of an undergraduate curriculum in radiology. Such consideration, however, is fundamentally dependent upon an initially well defined undergraduate teaching program in radiology.

The long-range objectives for the undergraduate curriculum in radiology should mirror the rapid growth of the field. In retrospect, the original area of radiology, encompassing radiographic techniques and interpretation, no longer fills the needs of this growth. The future undergraduate curriculum must, in addition, include instruction in the sister areas of radiation hygiene and radioisotope studies.

In future curriculum planning, one must be cognizant of two related factors: (1) the responsibility of the teacher to convey specialized knowledge and (2) the need to correlate this knowledge with teaching activities in other fields of dentistry. If these two factors are representative of the general objectives of the

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future curriculum in radiology, the element of establishing equitable clock-hour requirements within the total dental curriculum for teaching radiology becomes self-definitive.

This connotation implies that the future undergraduate curriculum in radiology should constitute a process of instruction coupled with continuous correlation over the entire four undergraduate years. Under such conditions, this program will tend to act as a stabilizing influence upon the total undergraduate dental curriculum. It will create, in effect, an area of basic clinical science which will help to blend basic science and clinical science instruction.

Specialized knowledge in radiology lends itself to a division into the previously mentioned categories of (1) radiographic techniques and interpretation, (2) radiation hygiene, and (3) radioisotope studies. Curriculum planning must be governed by a choice of subject matter in each of these categories reflecting "must-know," "should-know," and "nice-to-know" knowledge. The new curriculum should selectively concentrate upon the following:

1. Morphologic range of normality.
2. Biophysical principles in radiology (including radiographic techniques).
3. Incipiency of disease in bone and other oral tissues.
4. Characteristics of frank disease processes.
5. Analysis of growth and development; evaluation of disease and response to treatment.

Using these five objectives, I will attempt to propose the typical undergraduate radiology curriculum of the future.

The concept of "morphologic range of normality" will probably be best expressed in the formal courses in radiology taught during the second undergraduate year. The student at this level will have a growing knowledge of basic principles in histology, anatomy, biochemistry and physiology. However, there is every reason to predict that these radiology–basic science relationships will be initiated as early as the latter part of the first year, with the radiology teacher functioning as a consulting lecturer in selected basic science courses. For example, once the first-year student has been exposed to the fundamentals of dental histology and oral anatomy, a lecture discussing the relationship of the radiographic range of normality of the dentoalveolar unit to its histologic counterpart will provide a visual transition from the microscope to the patient. The ramifications of such a method in terms of training the student to apply basic science principles to clinical practice are self-evident.

In the curriculum of the future, a minimum of four courses in radiology will be taught during the second year. A course in radiographic anatomy of the head, utilizing closed-circuit television as a teaching aid, will ground the student in the interpretation of the normal radiographic morphology of the head. His knowledge of the osteology of the head acquired in the first-year courses in gross anatomy and oral anatomy will serve as prerequisites for this phase of radiology. In a second companion course in clinical radiography, the student will concentrate on mastering the techniques of extraoral radiography in an

attempt to establish a more realistic correlation between the clinical and didactic phases of radiographic anatomy of the head. The biophysicist, who will be a member of the dental radiology department of the future, will teach the course in radiation physics to the second-year students. This course will serve to orient the student in physical principles of radiation, fundamentals of radiation hygiene, and the use of radioisotopes in research and as an energy source for clinical radiography. A fourth course in radiology offered at the second-year level will introduce the student to the radiographic morphology of the teeth and supporting structures, with special emphasis upon the radiographic evaluation of the growth and development of the dentoalveolar unit. As this course draws to a close, it will achieve a final objective in developing an initial understanding of the radiographic evaluation of the incipiency of disease in bone and other oral tissues. At the end of the second year, the student will be well grounded in radiographic principles as they pertain to the range of normality. At this level of his training he will interpret disease states radiographically only in terms of their deviation from the normal.

During the third year the lecture course in radiographic interpretation of pathologic changes will demonstrate specific disease entities and orient the student in the application of radiographic interpretation to the differential diagnosis of these diseases. This course in radiology will be carefully phased with the course in oral pathology, permitting the radiologist and the pathologist to discuss specific diseases concurrently. The third-year student will again experience the transition from microscope to patient via the radiograph. However, in contrast to the expression of this discipline in the second year, he will be exposed to concepts of disease rather than those of normality.

Throughout the entire third year the student will be assigned to the radiology clinic for a minimum of three one-week clerkships. In the first of these sessions, he will focus his efforts primarily upon intraoral techniques. However, he will be responsible for all phases of radiography, darkroom techniques, and the practical application of radiation hygiene. During the week of the third clerkship, the student will receive instruction in cephalometry and the utilization of radiopaque substances in the radiographic diagnosis of salivary gland disease and cystic cavity and paranasal sinus involvements.

Having focused his attention upon the radiographic evaluation of normality, incipiency of disease and frank disease processes during the first three years of dental school, the fourth-year student will be prepared for the application of this knowledge to clinical practice. The fourth-year lecture courses in radiology will be designed to (1) stress the utilization of the radiographic service in comprehensive oral diagnosis, (2) re-evaluate dental x-ray equipment and darkroom procedures, and (3) discuss practical methods for implementing radiation hygiene in dental practice.

A radiology seminar course at the fourth-year level will function as a medium for complete oral radiographic evaluation of the patient. Each fourth-year student will be required to do a complete radiographic work-up on a minimum of two patients. The work-up will include posteroanterior, Waters', and

lateral jaw projections in addition to periapical, bitewing, and occlusal radiography. Where indicated, the student will be required to work with cephalometry, sialography, and other types of special films. The student will prepare each case for seminar presentation. In combination, the two fourth-year courses in radiology will stress the application of radiographic methods in the analysis of growth and development and in the evaluation of disease processes and the response of the patient to completed dental treatment.

An attempt has been made to propose a general plan for a future undergraduate curriculum in radiology comprising approximately 100 clock hours. The desirability of such a curriculum is based upon the rapid expansion of the field of radiology in dentistry.

STEREOSCOPIC ROENTGENOGRAPHY IN DENTISTRY

A Direct Method of Examining Stereoroentgenograms

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THE present aim of dental roentgenology is to obtain, by all available techniques, the best possible examination of dental structures. The long-cone technique, fast roentgenographic films, and improved x-ray apparatus show that dentists are trying to obtain better roentgenograms and more accurate interpretations from their roentgenographic examinations. Stereoscopic roentgenography can also contribute to the improvement of dental x-ray examinations. This improvement can occur not only in roentgenographic localization, where it undoubtedly finds its greatest use, but also in the examination of bony structures where there is any doubt as to the interpretation of the roentgenographic shadows. Using this technique, one is able to see the structure in three dimensions and thus with a perfection that cannot be equalled by any other roentgenographic means. Stereoscopy gives a perception of depth whenever retinal elements situated in the individual eyes are stimulated by the individual images on two roentgenograms taken with a horizontal shift. These images consequently unite to produce a single visual sensation. Differences produced by a vertical shift do not produce the stereoscopic effect.

HISTORY

One of the first dentists to apply stereoscopic techniques to dental roentgenography was Edmund Kelles who started to study the method in 1903 and who, in 1912, published an extensive article expounding the fundamentals of his method of dental stereoroentgenography. In 1924 and 1932 LeMaster[1] published articles concerning the application of stereoscopy in dentistry which, in turn, contributed greatly to the development of this method of roentgenologic examination. Wald[2] (1929 and 1936), Hodgson[3] (1927), Johnson[4] (1930), Thompson[5] (1936), Ennis[6] (1939), Berghagen[7] (1951), and Mazzoni (1954) also contributed greatly to improvement of the technique while indicating the value of the stereoscopic method of examination. Professor Contreras of the University of Chile is an enthusiastic supporter of this technique and presents lectures on the subject at the Faculty of Odontology.

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TECHNIQUE

The stereoroentgenographic examination gives the examiner the impression of perspective or depth; it is the ideal method for the localization of teeth, foreign bodies, or other abnormalities in the jaws. It also provides more accurate visualization of dental structures. Stereoscopy presents certain difficulties, which most probably explain why the method has not become more popular in dental circles around the world. However, by presenting a correct technique to be strictly followed, we shall attempt to make it simple and within the reach of all roentgenologists.

In this examination one has to obtain two roentgenograms of the same region, one film for the observer's right eye and another for his left eye. The patient's head must be held steady, and the film must be placed exactly in the same spot in both exposures.

Fig. 1.

Fig. 2.

Fig. 1.—The two positions of the x-ray tube for stereoscopic roentgenography of a supernumerary tooth.

Fig. 2.—Diagrammatic representation of the stereoroentgenographic result of an examination of a supernumerary tooth. Through fusion of the images, the extra tooth can be seen to be on the vestibular side.

Film Placing.—Since two roentgenograms of the same region are required, and since both films have to be placed in exactly the same position, a method is needed by which this condition can be obtained easily. A small quantity of warm impression compound is put on a "chassis," such as the corner of an envelop (the initial film is placed in the envelope corner), and an impression of the region is taken. This impression facilitates the removal of the film and the repetition of as many exposures as desired, with the film always in the same position. This method is described in more detail in a previous publication.[8]

The Shifting of the X-ray Tube.—The shifting of the x-ray tube in both directions can be achieved by different methods. Some use special apparatus,

while others use mathematical calculations or both. We usually shift the tube to the right and left of the region to be examined, in the horizontal plane. The equivalent of 5 or 6 degrees to the right side and 5 degrees to the left side is utilized (Fig. 1).

Fig. 2 is a diagrammatic representation of the result obtained and can be examined by means direct stereoscopic examination.

Stereoroentgenographic Film.—Whenever the films are removed, the packets should be marked* with the letters *R* and *L*, respectively. After developing, fixing, washing, and drying, the films must be marked so that they can be identified.

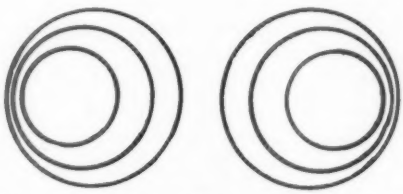


Fig. 3.—Circles demonstrating fusion of the images. The fusion of two figures is accomplished by placing a forefinger exactly in the center and at a short distance from the circles; subsequent focusing of the eyes on the finger tip enables the observer to see a cone, with the larger circle being nearer, giving the impression of a third dimension. This is fusion through convergence.

Apparatus Used for Examination of Stereo Films.—When the films are processed and identified, they are placed in the stereoscopic viewer. This is an optical instrument with which the films are studied. Here, with a little practice, the two images can be fused by the examiner, and a sensation of perspective or depth can be achieved. Several kinds of apparatus may be used for this examination. In our opinion, the Ritter intraoral stereoscopic viewer is the most efficient one available. Stereoscopic viewers used for viewing colored stereo slides can also be used, but these call for a small amount of adaptation.[8] The position of each film must be correct; the film taken on the right side must be placed on the right side of the apparatus, and the film taken on the left side must be placed on the corresponding side of the apparatus.

Direct Method of Examining Stereoroentgenograms.—We have had great difficulty in locating stereoscopic apparatus since we became interested in teaching the stereoroentgenographic method in the Department of Roentgenology at the University of São Paulo and to professional dentists.

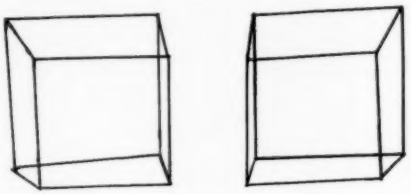
Ritter's apparatus is no longer manufactured, and a similar apparatus is not commercially available. Such apparatus is not to be found easily. Consequently, we tried direct examination while searching for a solution to the problem. This was first done with stereoscopic pictures and then with stereoroentgenograms. Fusion of the two images is accomplished with some practice and patience; the perception of depth is subsequently seen without the help of a stereoscopic viewer. Fusion of images can be accomplished through

*The authors use a lead letter *R* or *L* on the film during the x-ray exposure to facilitate identification.

convergence or divergence of the eyes. Young persons find it easier to fuse the images through convergence. Older persons fuse them better and more easily through divergence.

Fusion of images through convergence can be practiced by observing Fig. 3. Put a forefinger halfway between the circles and focus your eyse on it. With a little effort, fusion is accomplished; now take the finger away and focus on the circles; note that the larger circle is nearer and the smaller one farther away.

Fig. 5.



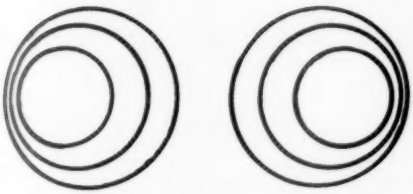


Fig. 4.

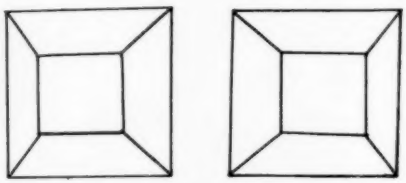


Fig. 6.

Fig. 4.—Circles that also facilitate the study of direct fusion of stereoscopic images. Fusion of the circles gives an image of a cone, with the smaller circle nearer and the larger one farther away. This is what happens when fusion is effected through visual convergence. When fusion through divergence is used, one sees the larger circle as being nearer.

Fig. 5.—Stereoscopic diagrams for practicing the direct method of viewing stereoroentgenograms. A cube is seen.

Fig. 6.—Stereoscopic figures representing the trunk of a pyramid which can be used for sight practice to facilitate the direct-vision stereoscopic method.

On the other hand, when one observes Fig. 3 by trying to focus it as far away as possible or by trying to relax the eyes, the images appear to be in the center of the figure. In this way the smaller circle on the figure will be nearer and the larger one farther away. This is fusion through divergence.

It should be noted that visual fusion accomplished through divergence produces an image with true perspective and is equivalent to the one seen in a stereoscopic viewer. This is explained by the fact that when the pictures are viewed through near vision or by shortsighted persons, crossed diplopia is used. In other words, the right eye sees the left-hand picture, and the left eye sees the right-hand picture. When the pictures are viewed from a distance, only diplopia occurs, and each eye sees its respective picture.

Practice with Figs. 3, 4, 5, and 6 will help one to master the direct method of examination of stereoscopic roentgenograms. This is an important step in the use of this roentgenographic method.

Those who use fusion by convergence will see the position of teeth or pathologic lesions in inverse positions. For example, when stereoroentgenography is used on an impacted canine, and the viewer is looking from the

Fig. 7.

Fig. 8.

Fig. 7.—Stereoscopic pictures in which the perception of depth can be achieved through the direct method.

Fig. 8.—Stereoscopic pictures with which the reader can also observe the phenomenon of a third dimension without the use of a stereoscopic viewer.

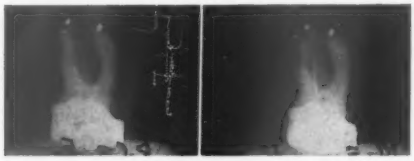


Fig. 9.—Stereoscopic roentgenograms of a molar tooth in which stereoscopic vision will be more easily attained through direct vision.

palatal side, if the canine should be nearer it will appear to be placed on the vestibular side. Fusion through divergence gives the true position of the

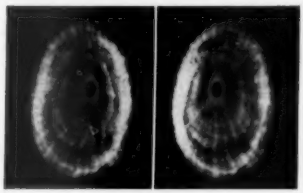


Fig. 10.—Stereoscopic roentgenograms of a shell. A volcano-like crater will be seen upon fusion of the images.

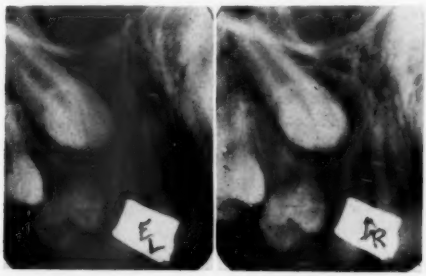


Fig. 11.—Stereo films of the upper cuspid region. The impacted tooth can be localized on the vestibular side in relation to the roots of the incisors. A piece of lead placed in the hot compound can also be observed.

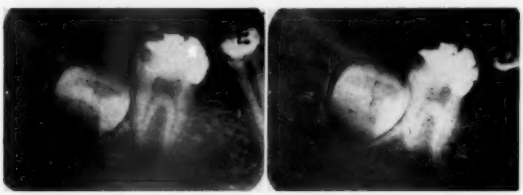


Fig. 12.—Stereoroentgenograms of a lower third molar as seen from the lingual side, demonstrating its buccal position.

Fig 13.—An impacted upper canine as seen from the palatal side. The tooth can be observed as being nearer than the lateral incisor. This means that it is situated palatally.

tooth. The photographs shown in Figs. 7 and 8 are useful for practicing one's vision in the perception of stereoscopic phenomena. This is recommended after one has practiced with Figs. 3 to 6.

After some practice, one is able to obtain wonderful stereoscopic vision from photographs. Stereoscopic roentgenograms, as shown in Figs. 9 and 10 are very helpful in practicing this method. These figures help the viewer to obtain faster stereoscopic vision.

The roentgenograms shown in Fig. 9 represent a stereoscopic roentgenographic examination of an extracted molar tooth. Fig. 10 shows roentgenograms made of a sea shell. The roentgenograms shown in Figs. 11 to 17 demonstrate

Fig. 14.

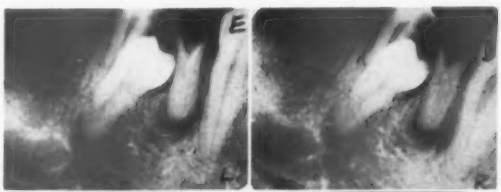


Fig. 15.

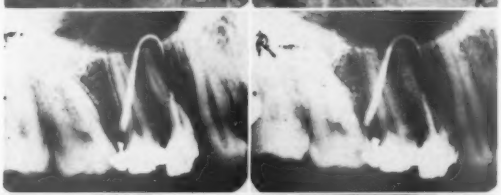


Fig. 16.

Fig. 17.

Fig. 14.—Stereoscopic roentgenograms of the lower premolar area. The supernumerary premolar is perceived through stereoscopic vision as being situated close to the lingual side. The importance of this method in the perfect localization of impacted teeth is demonstrated beyond any reasonable doubt. No other radiologic technique can be compared with it, so far as precision and speed are concerned.

Fig. 15.—Stereoscopic roentgenograms of the upper premolar region seen from the vestibular side. Note that a cone of gutta-percha placed in a sinus tract located on the vestibular side moves to the region of the palatine root of the first premolar. The end of the cone is clearly seen to be on the vestibular side.

Fig. 16.—Stereoscopic roentgenograms of the upper premolar region showing another example of a sinus tract as seen from the vestibular side.

Fig. 17.—Stereoscopic roentgenograms of the lower molar region where two impacted teeth can be observed. Through direct stereoscopic vision the impacted second molar can be seen to be on the lingual side and the third molar on the vestibular side. This case proves the value of stereoscopic examination in perfect localization, even in the mandibular region where some authors do not advise its use.

clinical cases in which this method rendered outstanding service to the oral surgeon and endodontist from the standpoint of perfect localization of impacted teeth and/or the study of periapical tissues.

SUMMARY

We have presented a limited study of stereoscopic roentgenography as applied to dentistry. We have discussed its technique and have described in particular a method of studying stereoroentgenograms by direct examination. Fusion of the images is accomplished without the help of a stereoscopic viewer. We feel that this technique will certainly help in popularizing stereoscopic roentgenography.

CONCLUSIONS

1. Done by the method described, direct stereoscopic examination will not present any difficulty for the dentist.

2. Stereoscopic roentgenography is an ideal method, as well as a more accurate one, for localizing the positions of teeth, tumors, and other abnormalities in the jaws.

3. Stereoscopic roentgenography is a more accurate method for the detailed study of small maxillary structures; by this method, one is able to see these structures in three dimensions.

4. In endodontics, stereoroentgenography is clearly indicated—not only to confirm the position of the lesion but also to find its relationship to important structures, such as the maxillary sinus, the mandibular canal, etc.

5. The direct method of perceiving the stereoscopic phenomenon greatly facilitates the application of stereoroentgenography to dentistry in spite of the fact that the technique is somewhat difficult to master at the beginning.

6. We sincerely believe that, through the popularization of this method of direct examination, stereoscopy will one day occupy a prominent place in dental roentgenology.

EDITOR'S NOTE

It must be noted that there are two ways in which the images fuse into a single picture when the proposed direct-viewing method is used. Fusion by divergence gives the correct perspective, whereas fusion through convergence gives us a three-dimensional image that is "inside out" or, in other words, as if we were looking at the object from behind it. The elimination of fusion through convergence can be accomplished by placing a piece of paper (edgewise) between the eyes and the pictures, so that each eye can see only its respective picture. (The editor is indebted to Dr. L. R. Manson-Hing for his editorial assistance.)

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ROENTGENO-QUESTIONS

Will the parallel placement of film aid in getting better roentgenograms even though the extension cone is not used?

Basically, with parallel film placement the long extension cone gives roentgenograms of better quality than can be obtained with the short cone. Because of magnification, it would be best not to use parallel film placement without an extension cone as a routine procedure. There are certain areas in the mouth, however, where one might like to eliminate superimposition of some structures that are interfering with the interpretation of others. The most common of these is the upper periapical molar area. Even though some magnification will occur, the apices of upper molar teeth will be more clearly visualized and in some instances the zygomatic shadow can be eliminated from the roentgenogram. The alveolar crest of the ridge and the bone trabeculae appear to be more normally positioned in most areas, even with the magnification, when the parallel film placement is used. The lower molar region with the close tooth-film relationship also appears to benefit from this placement. With an accurate bisecting angle technique, the occlusal-apical lengths of teeth are more correct; however, parallel film placement is useful in regions where reproduction of normal anatomic relationships, even with magnification, would be helpful in diagnosis.

The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

ROENTGENO-ODDITIES

The "Submerged" Tooth

THE accompanying roentgenogram reveals a tooth crown that contains a metallic restoration completely embedded in maxillary bone between the apices of the maxillary second premolar and second molar. This tooth was observed during the course of a routine examination of a 19-year-old patient. There was no history of pain or discomfort in the region in question, and all teeth responded normally to the electric pulp tester and to percussion. Upon removal, the retained tooth was found to be ankylosed. A draining sinus tract was observed in the buccal alveolar mucosa near the apical region of the maxillary right second premolar.

The sinus views, both posteroanterior and lateral, indicated that the tooth was contained completely within the maxillary bone with no evidence of maxillary sinus involvement.

Gross examination indicated that the specimen consisted of the crown of a small upper molar tooth, resembling a first molar, with a small occlusal amalgam restoration and two irregular, flattened pieces of firm, brownish white to dark brown soft tissue measuring 1.7 by 1.7 by 0.4 cm. and 1.2 by 0.9 by 0.3 cm. Microscopic examination revealed irregular sections of fibrous connective tissue lined along one surface by loosely arranged hyperplastic stratified squamous epithelium. The connective tissue immediately adjacent to the epithelium was young and vascular and heavily infiltrated with lymphocytes, plasma cells, and macrophages. Distal to this area, the connective tissue assumed a more mature appearance and contained only scattered foci of chronic inflammatory cells. Along the periphery of the specimen, a few spicules of vital bone were seen. The soft tissue was consistent with the lining of the sinus occupied by the dislocated tooth. "The histopathologic diagnosis was described for the soft tissue as the lining of a traumatic sinus in the maxilla."

Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

Discussion.—These so-called "descending teeth" do not actually descend. They are found more often in the mandible than in the maxilla. For some reason, the tooth (more frequently a deciduous tooth) becomes ankylosed during the process of growth. As a result, the developing bone grows around the ankylosed tooth and the normal teeth erupt in their usual time sequence. Subsequently, the root of the ankylosed tooth becomes resorbed and the tooth crown,

which may be in any of various stages of resorption, is left embedded in bone. In this particular instance, the phenomenon which resulted in ankylosis apparently also developed a fistulous tract and an encapsulating membrane which was cystic in nature.

Jay D. Shaw, Commander (DC) USN
Homer S. Samuels, Lieutenant Commander (DC) USN
FPO, New York, New York

ORAL PATHOLOGY

General Section

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A HISTOCHEMICAL STUDY OF THE KERATOTIC PROCESS IN ORAL LESIONS DIAGNOSED CLINICALLY AS LEUKOPLAKIA

Samuel Turesky, D.M.D., A.B., Irving Glickman, D.M.D., B.S., and Jacqueline Provost, A.B., Boston, Mass.

STUDIES of epidermis and of dermal appendages identify sulfhydryl and disulfide groups and glycogen with physiologic keratinization.[1-9] The distribution of these chemical groups has also been studied in keratinized gingiva.[10-13] We were interested in obtaining information regarding the nature of pathologic keratinization (keratosis) as it occurs in human oral mucosa. For this purpose, a microscopic and histochemical study was made of eighty oral lesions diagnosed clinically as leukoplakia.

Biopsy specimens of the lesions were fixed either in 10 per cent formalin or in Bouin's solution, prepared in serial sections, and stained as follows: hematoxylin and eosin, Barrnett-Seligman technique for protein-bound sulfhydryl and disulfide groups, periodic acid–Schiff (PAS) technique for the demonstration of glycogen and carbohydrate-protein complexes. In order of descending frequency, the lesions were located as follows: buccal mucosa (32), gingiva (18), hard and soft palate (17), lip (6), tongue (5), and floor of the mouth (2). Biopsy specimens of comparable regions served as controls.

At the time of biopsy all the lesions had been diagnosed as leukoplakia, solely on the basis of clinical features. We were concerned primarily with the keratotic process which produces white plaquelike lesions in the oral cavity and is commonly designated as leukoplakia rather than with the appropriateness of the term.

MICROSCOPIC FINDINGS

The lesions presented certain consistent microscopic features and variations, regardless of their location. In all specimens the stratum corneum was thickened

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Abstract presented at the thirty-seventh annual meeting of the International Association for Dental Research in San Francisco, California, March 19 to 22, 1959.

and presented hyperkeratosis or parakeratosis, alone or in combination. In 65 per cent of the specimens there was some degree of hyperplasia of the underlying epithelium, with acanthosis and elongated and usually broadened rete pegs. In the remainder of the specimens the epithelium beneath the surface layer appeared either unaltered (25 per cent) or atrophic (10 per cent). Parakeratosis was associated more often with epithelial hyperplasia than hyperkeratosis, and the thickness of the underlying epithelium was greater in the former than in the latter. Inflammation in the underlying connective tissue was seen in 55 per cent of the specimens. (This finding is comparable to that reported by Ronstrup.[14]) In many instances leukocytic invasion and edema of the epithelium were also observed.

The lesions presented similar microscopic features, regardless of whether they occurred in normally keratinized, parakeratinized, or nonkeratinized areas of the oral mucosa. There were significant microscopic and histochemical differences between the hyperkeratotic and parakeratotic lesions.

In hyperkeratosis there was a prominent multilayered keratohyaline zone. The cells in this zone underwent progressive changes (Figs. 1 and 2) in the direction of the stratum corneum. The basophilic keratohyaline granules in the cytoplasm became larger and stained more deeply. Scattered granules within the cells took on eosinophilic stain until the entire cell became an amorphous eosinophilic mass, often with spherical remnants of the nucleus retained within vacuolar spaces. The cell boundaries were progressively obscured as the surface was approached, resulting in a stratum corneum of multilayered, flattened strands.

The Barrnett-Seligman technique revealed that with the eosinophilic transformation of the keratohyaline cells there was a pronounced increase in the concentration of sulfhydryl groups (Figs. 3 and 4). This resulted in the production of a sulfhydryl-rich transitional zone between the surface stratum corneum and the underlying keratohyaline layer. The transitional zone was not visible in sections stained with hematoxylin and eosin. The sulfhydryl content of the stratum corneum was strikingly reduced in comparison with the deeply staining transitional zone immediately subjacent and was even less than that observed in the remainder of the underlying epithelium.

Serial sections reduced with thioglycollic acid indicated that the reduction in amount of sulfhydryls in the stratum corneum of the hyperkeratotic lesions was accompanied by a marked increase in disulfide content (Figs. 5 and 6).

In the lesions in which parakeratosis of the epithelium rather than hyperkeratosis was the principal change, there was either no keratohyaline zone or only the slightest suggestion of one (Fig. 7). With isolated exceptions, the prickle-cell layer blended with the parakeratotic stratum corneum without an intervening keratohyaline zone. Flattening and elongation of the cells and pyknosis of the nuclei accompanied by a change from basophilic to eosinophilic staining occurred in the transformation of the polyhedral cellular structure of the prickle-cell layer to a multilayered parakeratotic surface.

In the parakeratotic lesions, the Barrnett-Seligman technique revealed that the change from the basophilic staining of the prickle-cell layer to the eosino-

Fig. 1.

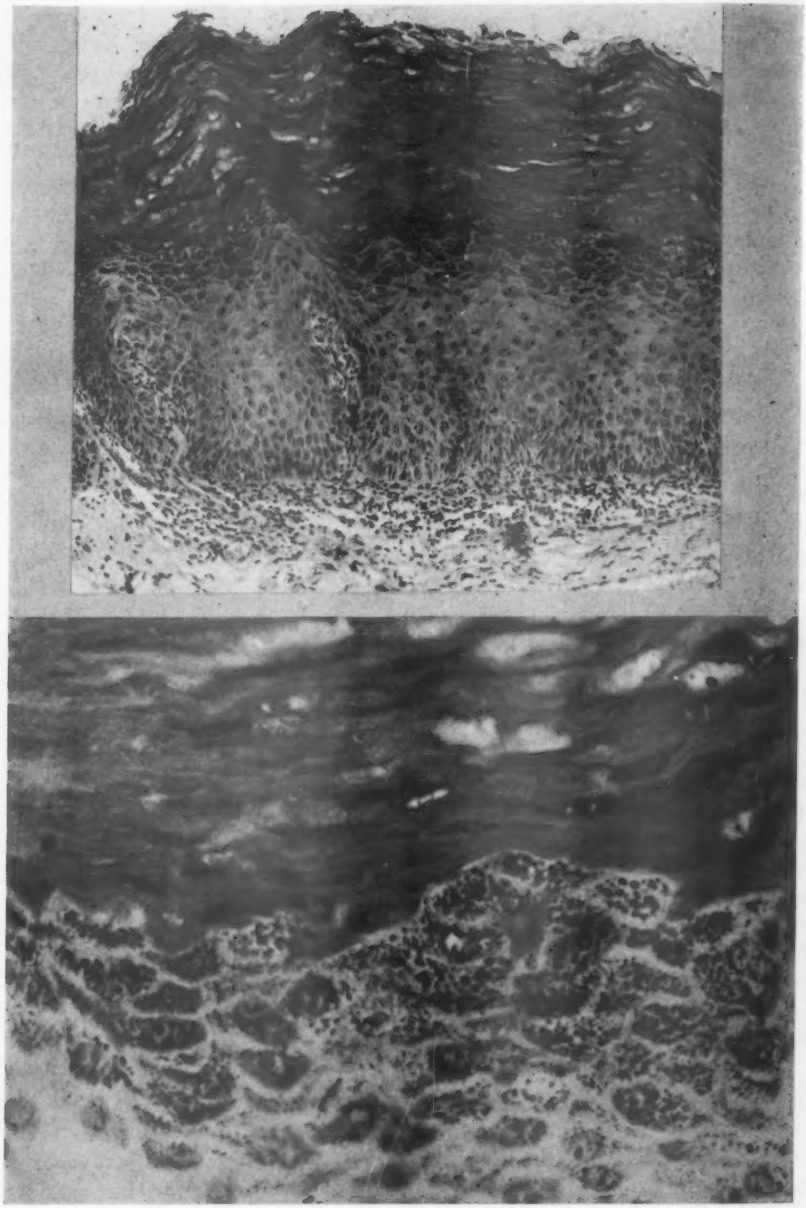
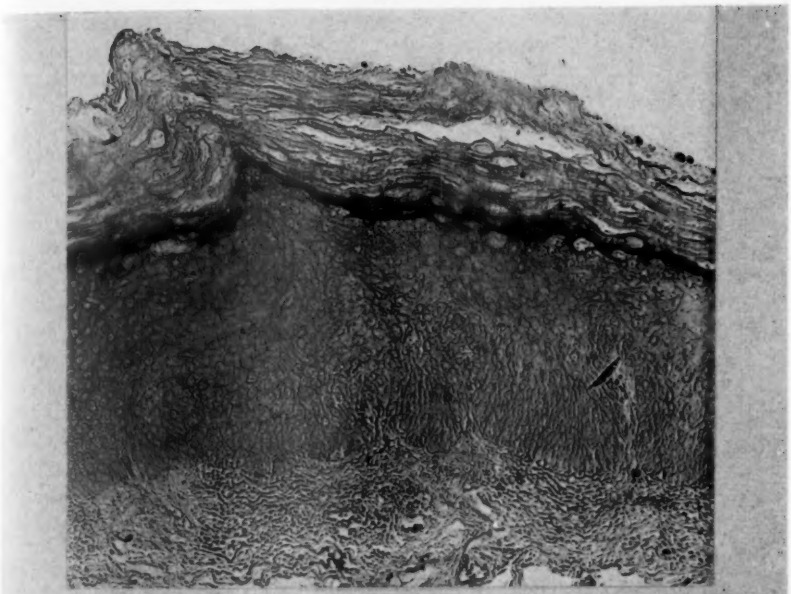


Fig. 2.

Fig. 1.—Hyperkeratotic lesion, buccal mucosa, with prominent keratohyaline layer and thickened stratum corneum. (Hematoxylin and eosin stain. Magnification, ×200; reduced ⅙.)

Fig. 2.—High-power view of lesion shown in Fig. 1, showing transformation of cells in keratohyaline layer to amorphous structure in stratum corneum. (Hematoxylin and eosin stain. Magnification, ×800; reduced ⅙.)

Fig. 3.



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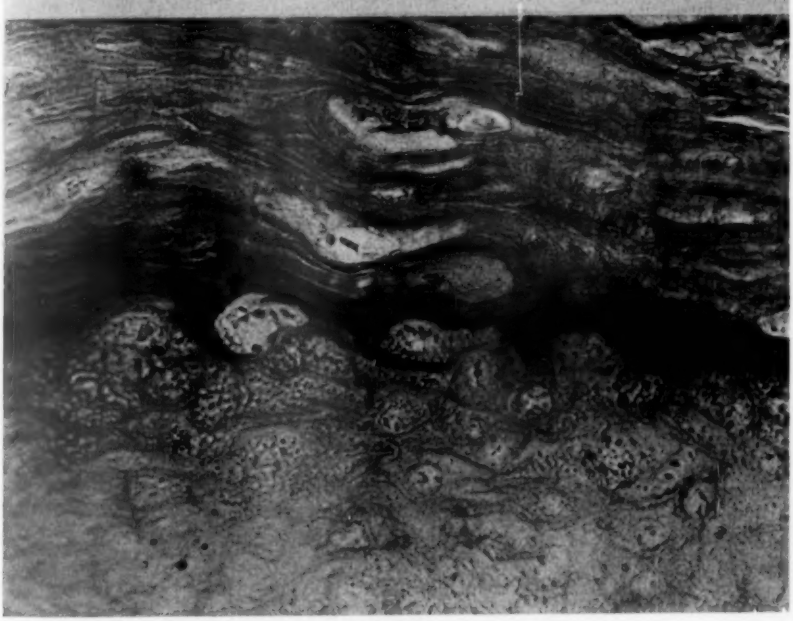
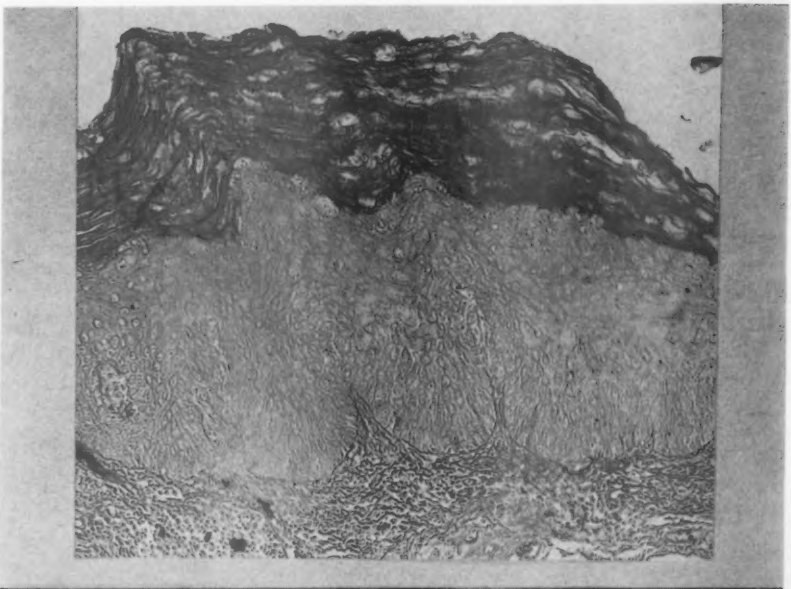


Fig. 3.—Serial section of tissue shown in Fig. 1, demonstrating distribution of sulfhydryl groups. Note the sulfhydryl-rich transitional zone subjacent to the corneum. The stratum corneum is almost devoid of sulfhydryls. (Barrnett-Seligman technique. Magnification, ×200; reduced ⅙.)

Fig. 4.—High-power view of section shown in Fig. 3. Note the sulfhydryl-rich "transitional" zone just above the keratohyaline layer. (Barrnett-Seligman technique. Magnification, ×800; reduced ⅙.)

Fig. 5.



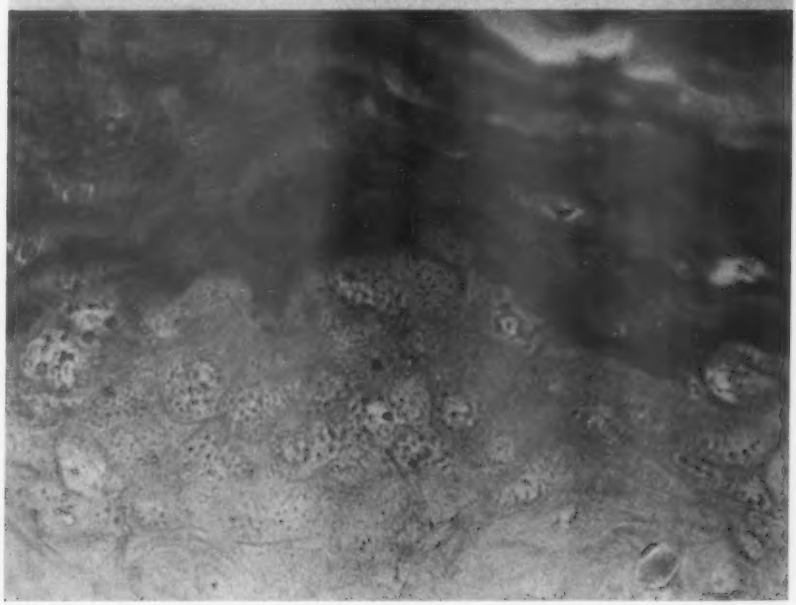


Fig. 6.

Fig. 5.—Serial section of lesion shown in Fig. 1, demonstrating concentration of disulfide linkages. Compare with Fig. 3. (Barrnett-Seligman technique. Magnification, ×200; reduced ⅓.)

Fig. 6.—High-power view of section shown in Fig. 5, showing concentration of disulfides in the stratum corneum immediately above the "transitional" zone. Compare with Fig. 4. (Barrnett-Seligman technique. Magnification, ×800; reduced ⅓.)

Fig. 7.

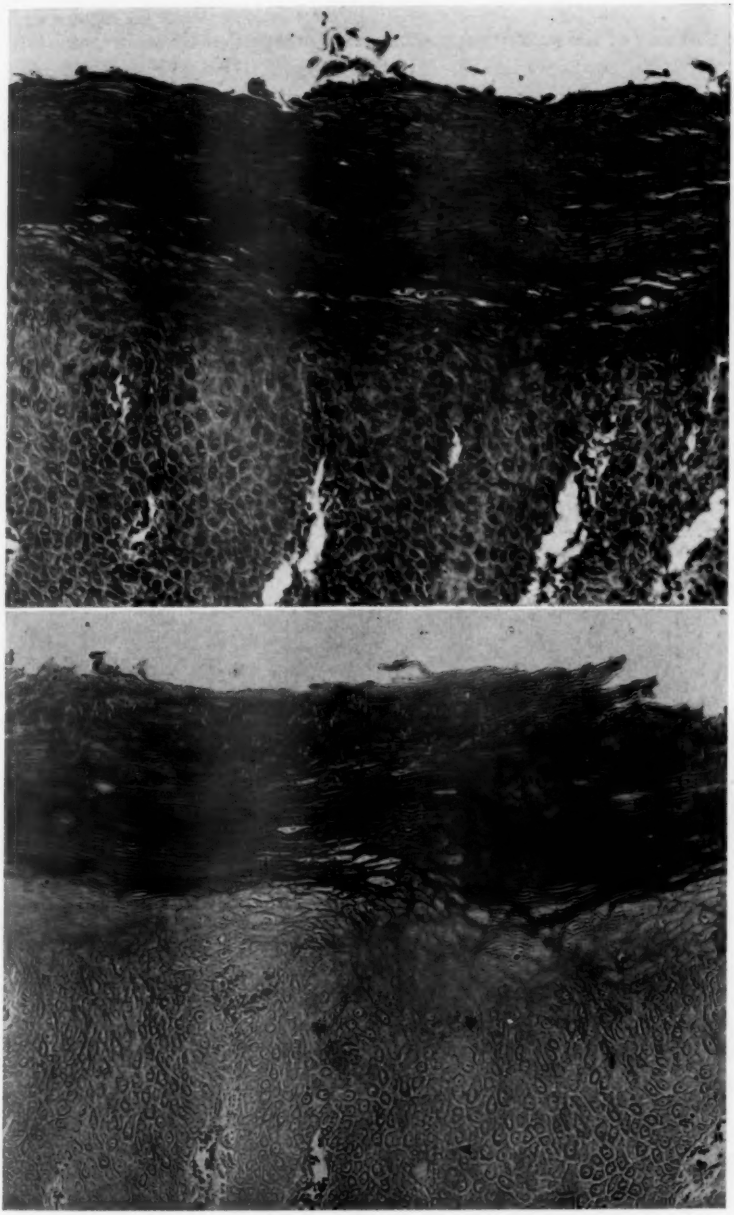


Fig. 8.

Fig. 7.—Parakeratotic lesion in the lip. Note absence of prominent keratohyaline layer. Compare with Fig. 1. (Hematoxylin and eosin stain. Magnification, ×200; reduced ⅓.)

Fig. 8.—Serial section of lesion shown in Fig. 7, showing concentration of sulfhydryls in the stratum corneum. (Barrnett-Seligman technique. Magnification, ×200; reduced ⅓.)

philic staining of the stratum corneum was accompanied by an increased staining of sulfhydryl groups (Fig. 8). In this respect, the stratum corneum of the parakeratotic lesions with an abundant distribution of sulfhydryl groups differed from the stratum corneum of the hyperkeratotic lesions in which sulfhydryls were essentially absent (Fig. 3). Reduction of the parakeratotic lesions with thioglycollic acid showed disulfide groups to be present in negligible amounts (Fig. 9). This also contrasted with the pronounced increase in disulfides in the stratum corneum of the hyperkeratotic lesions (Fig. 5).

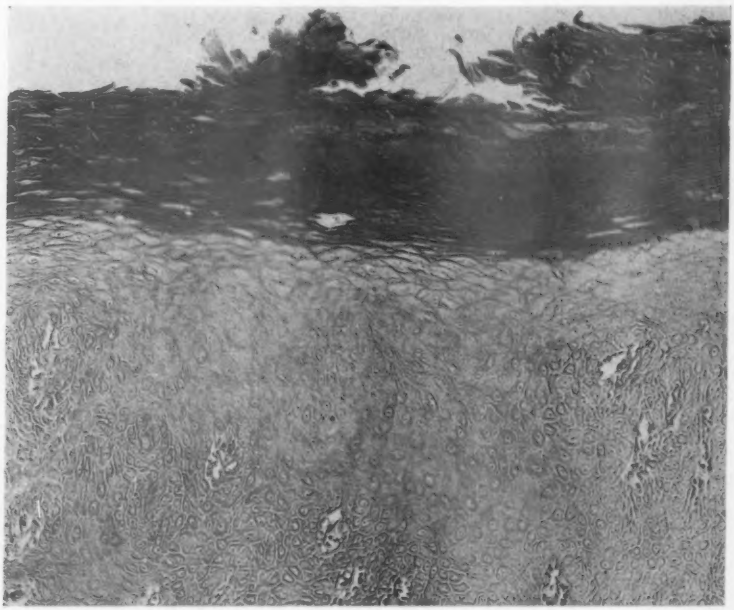


Fig. 9.—Serial section of specimen shown in Fig. 8, reduced with thioglycollic acid for detection of disulfide groups. Absence of change in intensity of stain when compared with Fig. 8 is indicative of negative finding. (Barrnett-Seligman technique. Magnification, ×200; reduced ⅓.)

Glycogen was present more frequently in the prickle cells and surface layers of the epithelium of parakeratotic lesions. It was seen only occasionally in the hyperkeratotic lesions (Figs. 10 and 11). The occurrence of glycogen appeared to be related to the degree of epithelial hyperplasia rather than to the nature of the surface changes. PAS-positive substances other than glycogen stained more deeply in the corneum of the parakeratotic lesions than in the hyperkeratotic lesions (Fig. 12).

In occasional specimens, the stratum corneum consisted of three or four alternate parakeratotic and hyperkeratotic bands. In some areas the parakeratotic band was at the surface (Fig. 13). In other areas of the same specimen the parakeratotic band appeared to have been shed, exposing the hyperkeratotic layer beneath it (Figs. 14 and 15). Staining for sulfhydryl indicated that

Fig. 10.

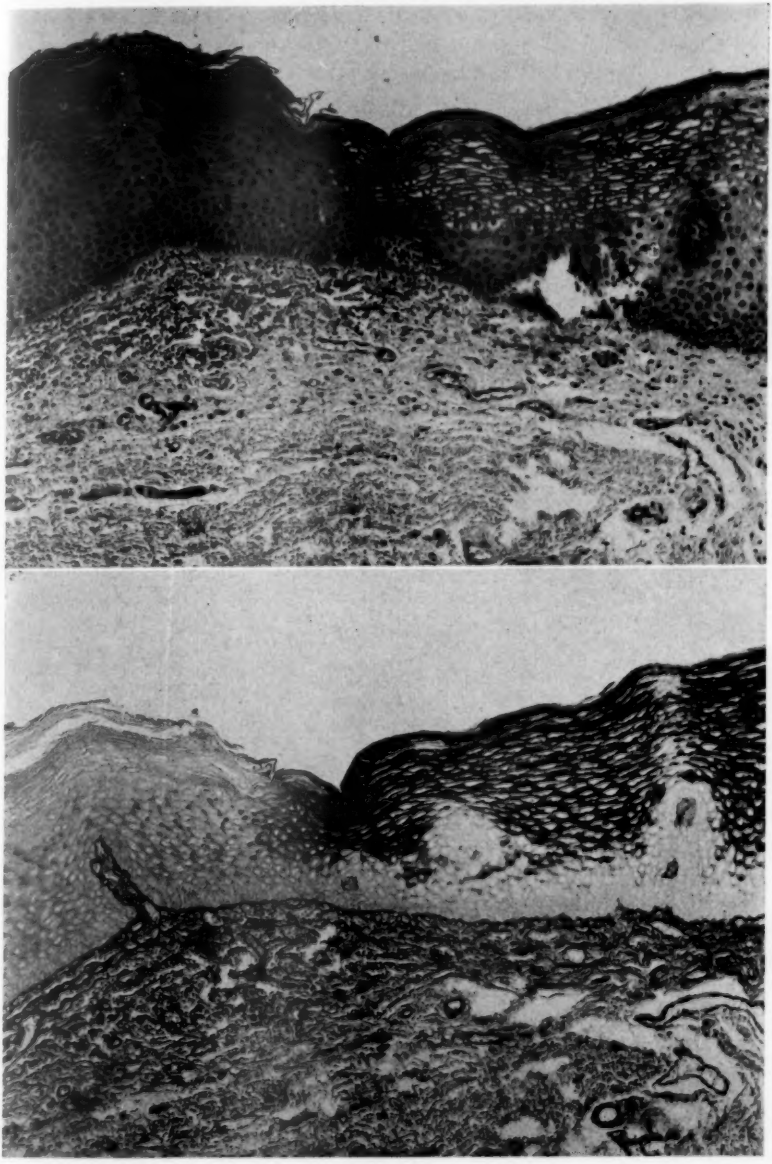


Fig. 11.

Fig. 10.—Lesion on soft palate, comparing area of hyperkeratosis (left) with bordering nonkeratinized epithelium. (Hematoxylin and eosin stain. Magnification, ×200; reduced ⅙.)

Fig. 11.—Serial section of lesion shown in Fig. 10, stained by the periodic acid–Schiff technique. Glycogen stains prominently only in the nonkeratinized epithelium (right). (Magnification, ×200; reduced ⅙.)

the bands differed histochemically. Sulfhydryls stained deeply in the bands which were parakeratotic but were barely detectable in those which were hyperkeratotic (Fig. 14). When reduced with thioglycollic acid, the staining of the hyperkeratotic bands was intensified, indicating the presence of disulfides. The staining of parakeratotic bands was not appreciably altered, which suggested a relative absence of disulfides (Fig. 15).

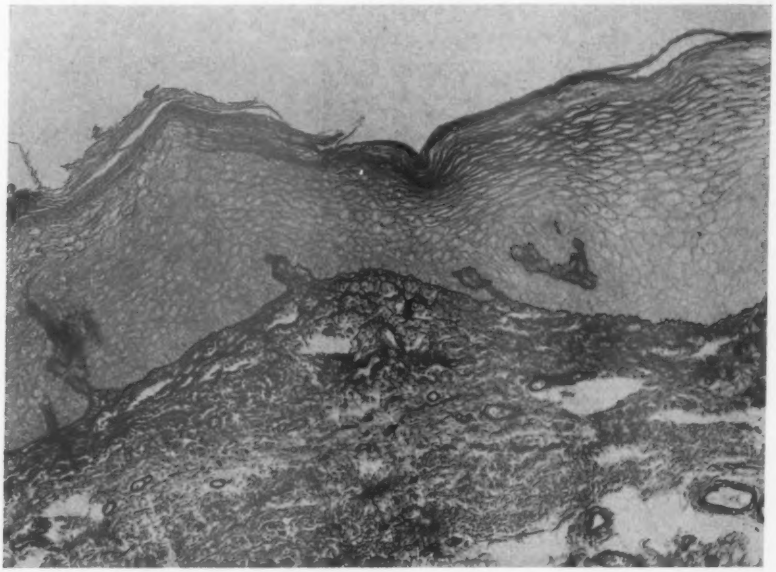


Fig. 12.—Serial section of specimen shown in Fig. 11, treated with diastase to remove glycogen prior to staining by the periodic acid–Schiff technique. Diastase-resistant substances are stained in the nonkeratinized epithelium (right) and only slightly in the hyperkeratotic lesion (left). (Magnification, ×200; reduced ⅙.)

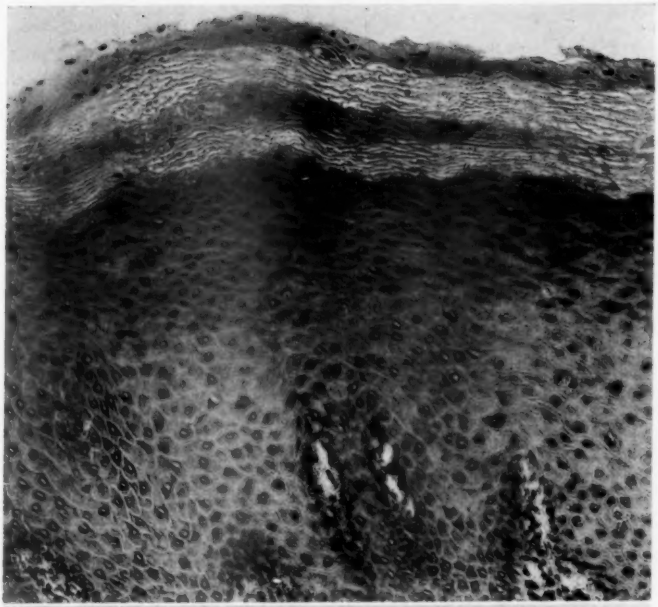
DISCUSSION

The study was concerned primarily with the keratotic process in lesions usually diagnosed clinically as leukoplakia. The findings confirm previous observations that thickening of the stratum corneum with either hyperkeratosis or parakeratosis, or both is a characteristic microscopic feature of such lesions. The lesions undergo similar keratotic changes, regardless of whether they occur in areas of the oral mucosa which are normally keratinized or nonkeratinized.

The Barrnett-Seligman technique revealed significant differences between the hyperkeratotic and parakeratotic lesions. It demonstrated that in hyperkeratosis there was a well-defined transitional zone between the keratohyaline layer and the stratum corneum which was not present in parakeratosis. This zone has been described in the literature[3] as a feature of so-called "hard" keratinization which occurs in such structures as nails or hair.

Hyperkeratotic and parakeratotic lesions also differed in the distribution of sulfhydryls and disulfides. In hyperkeratosis the sulfhydryl content was

Fig. 13.



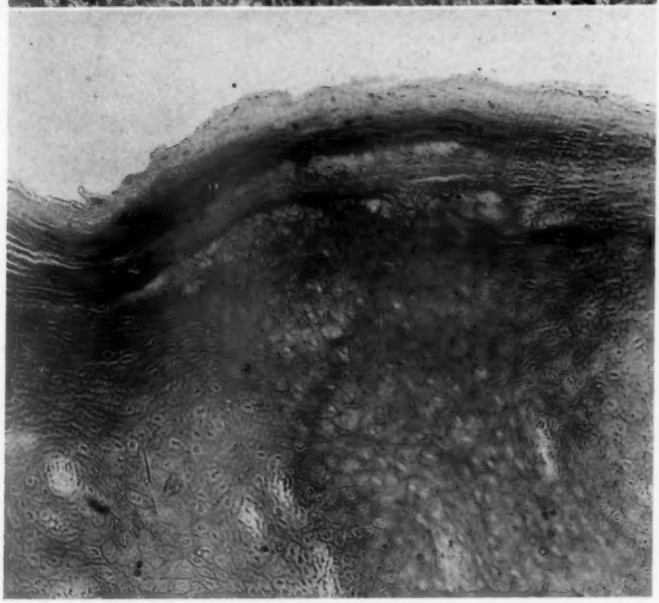


Fig. 14.

Fig. 13.—Lesion in buccal mucosa showing four alternate parakeratotic and hyperkeratotic bands in the stratum corneum. (Hematoxylin and eosin stain. Magnification, ×200; reduced ⅙.)

Fig. 14.—Serial section of lesion shown in Fig. 13, stained for sulfhydryls, showing three alternate bands in the stratum corneum. The surface parakeratotic layer shown in Fig. 13 is absent. Note the contrast between the dark stain of the parakeratotic band and the lighter stain of the hyperkeratotic bands above and below it. (Barrnett-Seligman technique. Magnification, ×200; reduced ⅙.)

greatest in the well-defined transitional zone. In the stratum corneum the sulfhydryl content was notably reduced in comparison with the transitional zone and was even less than in the Malpighian layer. In parakeratosis in which there was no well-defined transitional zone the greatest concentration of sulfhydryl occurred in the stratum corneum.

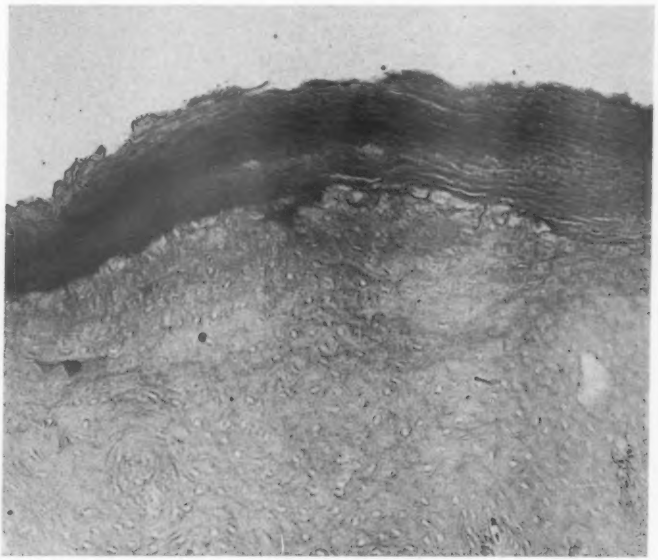


Fig. 15.—Serial section of specimen shown in Fig. 14, reduced with thioglycollic acid to reduce disulfides. Note that the hyperkeratotic bands which stained lightly in Fig. 14 take on a deeper stain. This is indicative of the presence of disulfides. The staining of the parakeratotic band is not appreciably altered. (Barrnett-Seligman technique. Magnification, ×200; reduced ⅕.)

The distribution of disulfide groups added meaning to the difference in sulfhydryl content of the hyperkeratotic and parakeratotic lesions. Disulfide was increased in the hyperkeratotic lesions where sulfhydryl content was reduced in the stratum corneum. In the parakeratotic lesions where sulfhydryls were increased in the stratum corneum, the disulfide content was negligible. It would appear, then, that a major difference between hyperkeratotic and parakeratotic lesions lies in the degree of oxidation of sulfhydryls to disulfides.

It is difficult to account for the structural and histochemical differences between the hyperkeratotic and parakeratotic lesions other than in terms of the rate of epithelial proliferation. In the parakeratotic lesions the over-all thickness of the epithelium was invariably greater and hyperplastic epithelial pegs occurred more frequently than in the hyperkeratotic lesions. Glycogen in the epithelium, interpreted as indicative of cellular activity,[5] was generally confined to the parakeratotic lesions. On the basis of these findings, we are inclined to

feel that in parakeratotic lesions the rapidity of cellular activity does not permit sufficient time for the structural and chemical differentiation required for hyperkeratosis.

The presence of alternating parakeratotic and hyperkeratotic bands in the stratum corneum of several lesions is of interest. It suggests that the process of hyperkeratosis may undergo modification, so that it occurs as an intermittent rather than a continuous process. When this occurs, hyperkeratotic bands are separated by less completely keratinized areas.

SUMMARY

The keratotic process was studied microscopically and histochemically in eighty oral lesions diagnosed clinically as leukoplakia. In all lesions the stratum corneum was thickened and presented varied degrees of hyperkeratosis or parakeratosis, separately or in combination. Several histochemical differences between the hyperkeratotic and parakeratotic lesions were noted. Of particular interest was the observation that hyperkeratotic lesions were characterized by an increase in disulfides and a diminution in sulfhydryls in the stratum corneum which did not occur in parakeratotic lesions. An intermittent type of keratosis was also described.

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GRANULAR-CELL MYOBLASTOMA OF THE ORAL CAVITY*

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THE granular-cell myoblastoma, since it was first described by Abrikossoff,[1] has been the source of much interest and conjecture. The diversity of synonyms used to describe this tumor ("Abrikossoff's tumor," "congenital epulis," "nonchromaffin paraganglioma," "granular-cell neurofibroma," "myoblastic myoma," "uniform myoblastoma," and "embryonal rhabdomyoblastoma") serves to stress the controversy regarding its nature.

This article presents a clinicopathologic study of seventeen patients with oral myoblastoma seen at the Mayo Clinic from 1907 to 1957. Tissue sections of these tumors were subjected to multiple staining techniques in an attempt to identify better the cytoplasmic constituents of the tumor cells and their relationship to the surrounding tissues in the hope of gaining further insight into the nature and derivation of these tumors.

GENERAL BACKGROUND

Although Abrikossoff is credited with publishing the first description of this entity in 1926, other authors[21, 27, 33] had reported tumors of a similar nature prior to that date. In Abrikossoff's five cases, all the lesions occurred in the tongue and were intimately associated with the striated muscle. It was suggested that the granular cells actually were degenerating adult striated-muscle fibers. Later, in 1931, Abrikossoff[2] modified this concept and, on the basis of work done by Godlewski[12] in 1902, suggested that the tumor cells were derived from myoblasts.

For a time, the theory of the myogenous origin of this tumor was accepted. In 1928 Klinge[17] reported two cases in which myoblastomas had arisen in areas normally devoid of striated muscle, and he proposed a theory of dysontogenesis as an explanation for this apparent incongruity. Schirmer[30] in 1932, Meyer[23] in 1932, and Klemperer[16] in 1934 denied the occurrence of any true transitions between the muscle fibers and the granular cells. Klemperer regarded these

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entities as neoplasms composed of myoblasts, while in 1937 Gray and Gruenfeld[13] considered these cells to be the products of necrobiotic changes in the muscle fibers.

In 1939 Leroux and Delarue[19] proposed an alternate theory which held that the granular cells were mesenchymal and of the histiocytic cell type. Other authors, notably Holle,[14] Ringertz,[29] Lauche,[18] and Wegelin[34] echoed this "storage-cell" concept.

In 1948 Fust and Custer[9] adopted a theory of neurogenesis. Such a theory, they felt, best explained the extraoral myoblastomas that were not associated with striated muscle. In 1949, after further study of their material, they concluded that both the lingual and extralingual tumors were neural in origin.[10] In 1952 Bangle[4] presented forty-three cases in which he had carried out varied histochemical investigations. Although he was unable to name a particular cell of origin, he favored the theory of neurogenesis.

On the basis of results of tissue culture, Murray[25] concluded in 1951 that cultures of these lesions bear a close resemblance to the cultures of normal and neoplastic striated muscle. In 1953 Stout[32] referred to this work and concurred with the hypothesis of myogenesis. He also noted a malignant variety of myoblastoma that is organoid in configuration and corresponds to the nonchromaffin paraganglioma described by Smetana and Scott[31] in 1951. Other examples of malignancy can be found in the literature, with von Meyenburg,[22] in 1929, reporting the first case. In 1955 Gamboa[11] reported a case of malignant granular-cell myoblastoma and reviewed thirty-two reported cases in the literature. Of these he could accept only ten as being valid representatives of a malignant myoblastoma. In none of the ten cases were the lesions located in the oral cavity.

According to Bissi and Magri,[5] who in 1955 made a survey of the literature and collected a total of 439 cases, the granular-cell myoblastoma can be found in practically every organ and tissue of the human body. The sites of 365 of these tumors could be determined definitely; 140 occurred in the tongue. Thirty-seven additional tumors were confined to the alveolus and were classified as congenital epulides. From the literature, we have been able to compile a list of 252 granular-cell myoblastomas of the oral cavity (including sixty-one congenital epulides). This would imply that in more than 50 per cent of the reported cases this lesion occurred in the oral cavity.

PRESENT STUDY

Materials and Methods.—From the tissue registry of the Mayo Clinic, we extracted sections of tissue from all tumors of the oral cavity surgically excised from 1907 through 1957 which had been diagnosed as granular-cell myoblastomas or were considered to have entered into the differential diagnosis. These consisted of 3,263 squamous-cell carcinomas of the mouth and tongue, 33 sarcomas of the tongue, and 239 benign lesions of the oral cavity, which included all varieties of epulides, peripheral giant-cell reparative granulomas, xanthomas,

TABLE I. ORAL MYOBLASTOMAS SEEN AT THE MAYO CLINIC FROM 1907 TO 1957 (16 CASES)

PATIENT	AGE (YEARS)	SEX	SIGNS AND SYMPTOMS*	DURATION	LOCATION	ORIGINAL DIAGNOSIS	SIZE	EPITHELIAL HYPERPLASIA	TREATMENT†	FOLLOW-UP (NO RECURRENCE AFTER YEARS NOTED)
1	36	F	Painless growth	1 week	Right side of tongue	Myoblastoma	—	–	Excision	12
2	54	M	White lesion	1 month	Left side of tongue	Myoblastoma	Small pea	+	Excision	3
3	46	F	Firm, indurated	6 months	Tip of tongue	Grade 1 squamous-cell epithelioma	0.8 × 0.7 × 0.5 cm.	+	Excision	6
4	38	M	Firm, flat	1 year	Dorsum of tongue	Myoblastoma	0.7 cm.	+	Excision	7
5	43	M	Multiple sites, slowly enlarged	3 years	(1) Left side, (2) Tip, (3) Right side } Tongue	Grade 4 squamous-cell epithelioma	(1) 0.8 cm. (2) 0.5 cm. (3) 2 × 3 cm.	+	Gland and node disks	—
6	31	M	Superficial growth	—	Dorsum of tongue	Grade 3 squamous-cell epithelioma	1 cm.	+	Excision and gland disks	—
7	41	M	Painless, hard mass	1 year	Left side of tongue	Myoblastoma	7 × 5 cm.	+	Excision	12
8	46	F	Firm nodule	3 weeks	Right side of tongue	Squamous-cell epithelioma	1.5 cm.	+	Excision	1
9	37	M	—	—	Tongue	Grade 2 squamous-cell epithelioma	—	+	Excision	30
10	52	F	Slightly tender nodule	4 months	Tip of tongue	Myoblastoma	1.4 × 1.3 cm.	+	Excision	—
11	52	M	Circular, hard, raised	4 months	Left side of tongue	Myoma (myoblastoma)	1.3 cm.	+	Excision	13
12	50	M	Small, gray ulcer	4 weeks	(Posterior third) Left side of tongue	Myoblastoma	0.5 cm.	–	Excision	2
13	19	F	Small nodule	—	Dorsum of tongue	Myoblastoma	1.0 × 0.5 × 0.3 cm.	–	Excision	—
14	62	M	Nodule	—	Tip of tongue	Myoblastoma	0.5 cm.	+	Excision	5
15	34	F	Nodule	—	Dorsum of tongue	Myoblastoma	0.6 cm.	+	Excision	—
16	34	F	Buttonlike nodule	—	Right side of tongue	Myoblastoma	0.5 cm.	+	Excision	3

*Signs and symptoms are impressions of the examiner.
†Excision refers to local removal of the tumor.

and rhabdomyomas. From this histologic survey, twelve cases of oral myoblastoma were added to the five cases previously reported in 1949 by Murphy, Dockerty, and Broders.[24]

The available gross specimens, which had been stored in a 10 per cent solution of buffered formalin, were reviewed and representative paraffin tissue blocks were made. From these blocks sections were cut and stained with or treated by the following media or methods: hematoxylin and eosin, Heidenhain's iron-hematoxylin stain, Mallory's phosphotungstic acid-hematoxylin, Sudan black B, periodic acid-Schiff reaction with and without diastase digestion, Weil's myelin-sheath stain, and the Bodian method for nerves.

Clinical Data.—The following observations are based on seventeen cases of histologically confirmed oral granular-cell myoblastomas. The clinical findings in these seventeen cases agreed essentially with those found in the medical and dental literature, and sixteen of the seventeen cases have been summarized in Table I.

Age and sex: The ages of sixteen of the seventeen patients at the time of operation ranged from 19 to 62 years, with an average age of 39.9 years and a median age of 41 years. In the seventeenth case the lesion presented as a congenital tumor in a newborn female infant. Distribution according to sex was of little significance; there were nine male patients and eight female patients.

Signs and symptoms: The most salient feature was a firm, asymptomatic, small nodule on the tongue. In fourteen cases this nodule was discovered by the patient, and in three cases it was an incidental finding at the time of physical examination. In one case a nodule in the tongue was noted to be associated with ulceration and episodic pain.

Location and size: Sixteen of the myoblastomas in this group were located in the substance of the tongue. Fifteen presented as single lesions, whereas in one case three separate lesions were noted. The lateral border was the predominant site (nine cases), with the tip and the dorsum being equally involved (four instances in each site). In only one instance was the posterior third portion of the lateral border involved. The diameters of the lesions ranged from 0.5 to 3 cm. In the case of multiple lesions, the tumors presented on the dorsum and lateral border. One tumor was located on the lower right alveolar ridge of the mandible of a newborn female infant (omitted from the table).

Duration: In only ten cases was the duration recorded. Duration was a matter of one week to three years. However, because of the asymptomatic nature of the nodules, the patients were, for the most part, vague in their recollections.

Pathologic Findings.—

Gross: All the available gross specimens appeared as small, firm nodules which lacked any distinctive characteristics. More often they were flat to slightly raised or buttonlike. The cut surface revealed a homogenous nodule which was white or brown-yellow. The lesions appeared sharply circumscribed on gross examination.

Microscopic: Routine staining with hematoxylin and eosin revealed the tumors in all cases to be of the uniform cell type, represented by large polygonal cells with faint or poorly visualized cytoplasmic membranes. The appearance of the cytoplasmic pink-staining granules was distinctive and consistent in the tumor cells. The granules varied in size and degree of aggregation. Most commonly, the granules were fine and were evenly distributed throughout the cytoplasm, while the coarse granules were seen to be round or irregular and often were found in clumps. The latter phenomenon gave the appearance of vacuolation of the cytoplasm (Fig. 1).

The nuclei lacked any distinguishing characteristics. They varied somewhat in size and shape and often were hyperchromatic, although they never assumed characteristics that would suggest malignancy. The nucleoli were small.

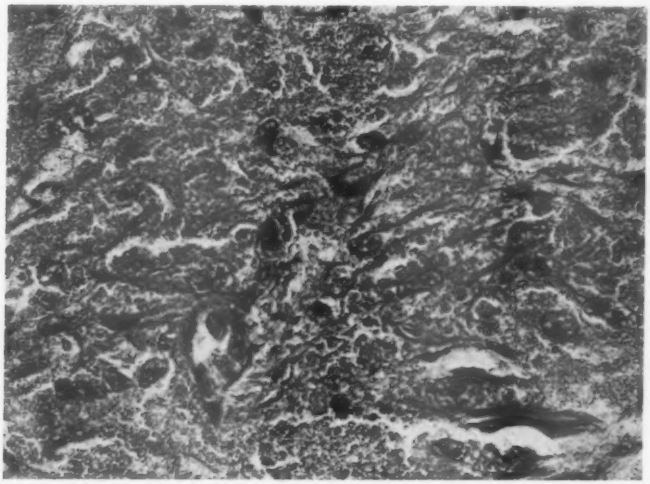

Fig. 1.—Representative granular cells (Case 12) that appear in a syncytial configuration. The granules vary in size and degree of aggregation and are surrounded by indistinct cytoplasmic membranes. (Hematoxylin and eosin stain. Magnification, ×500.)

No mitotic figures were observed in the microscopic preparations of the seventeen tumors. The fascicles and cords of granular cells frequently had the appearance of a syncytium. The individual tumor cells were surrounded by a distinct reticulin sheath. In certain instances delicate fibrils from this membrane tended to cleave the syncytium, giving the impression of compartments within the cytoplasmic mass.

Microscopic examination disclosed no evidence of encapsulation, although the circumscribed nature of the lesion was readily demonstrated. The tumor cells interdigitated into the adjacent fibrous stroma, striated muscle, and surrounded nerves, and often they were closely associated with the overlying squamous epithelium.

In all lesions of the tongue, striated muscle was intimately associated with the tumor cells. Cords of cells were interposed between strands of striated

muscle and, under the low-power objective, appeared as fingerlike projections which, upon casual observation, gave the appearance of a transition between the granular cells and the more differentiated sarcolemmal syncytium. Striations of a myofibrillar nature were totally absent in the tumor cells. In some instances the fine reticular fibers coursing through a cell syncytium gave the false impression of cross striations. The fibrous stroma of the tumor was not distinctive. It appeared to be a passive component of the tumor and to have been derived from the supporting elements of the infiltrated submucosa and striated muscles.

Nerves were found in association with the tumor in all cases. The most frequent pattern was one in which the tumor cells were concentrically arranged

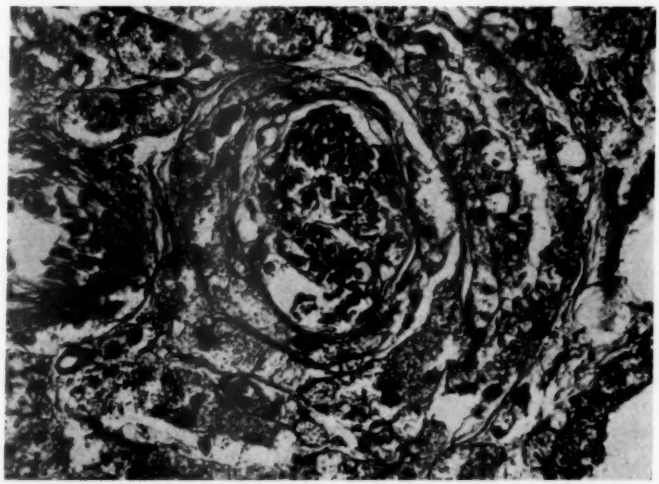

Fig. 2.—Tumor cells (Case 4) within the structure of the perineurium, whereas the endoneurium is intact. The surrounding tumor cells have reverted to a whorling pattern of growth. (Hematoxylin and eosin stain. Magnification, ×300.)

about a myelinated nerve. In only one microscopic section were tumor cells found within the structure of a nerve. In this instance the perineurium of a small nerve within the tumor mass had been invaded. The endoneurium was seen to be intact (Fig. 2), and there was no increase in the number of Schwann cells. There was no histologic evidence to suggest that any component of the nerve was the source of the granular cells. Involvement of nerves would appear to be secondary to the infiltrative growth of the tumor cells. Nerves distant from the aggregate of tumor cells were not involved.

In thirteen of the seventeen cases there were hyperplastic changes of the overlying squamous epithelium (Fig. 3). The proximity of the tumor cells to the surface epithelium did not appear to be responsible for this epithelial hyperplasia, for in the four cases in which there were no hyperplastic changes the tumor cells were in contact with the basement membrane. Squamous-cell carcinoma was not encountered in association with the granular-cell myoblastoma

in a single case in this series, although in five cases the lesions had previously been erroneously diagnosed as squamous-cell carcinoma.

The one case of a congenital myoblastoma on the lower alveolus of a newborn female infant was studied. Examination of serial sections of the lesion did not demonstrate epithelial inclusions or striated muscle within the substance

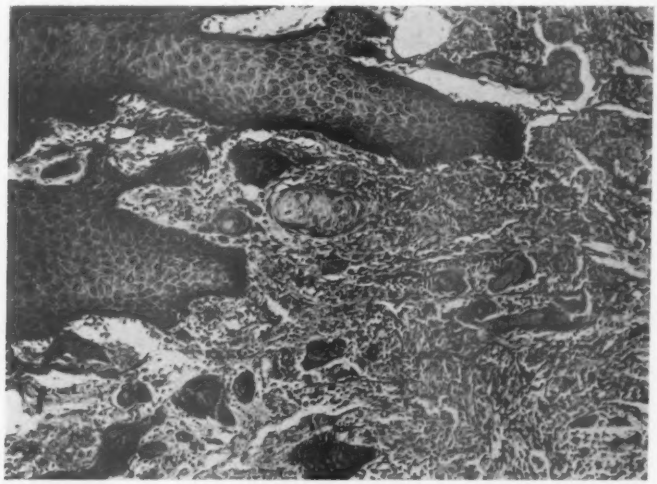

Fig. 3.—A downgrowth (Case 9) of the overlying epithelium with islands of squamous cells and pearl formation. This frequent finding in the oral myoblastomas often led to a misdiagnosis of squamous-cell carcinoma. (Hematoxylin and eosin stain. Magnification, ×80.)

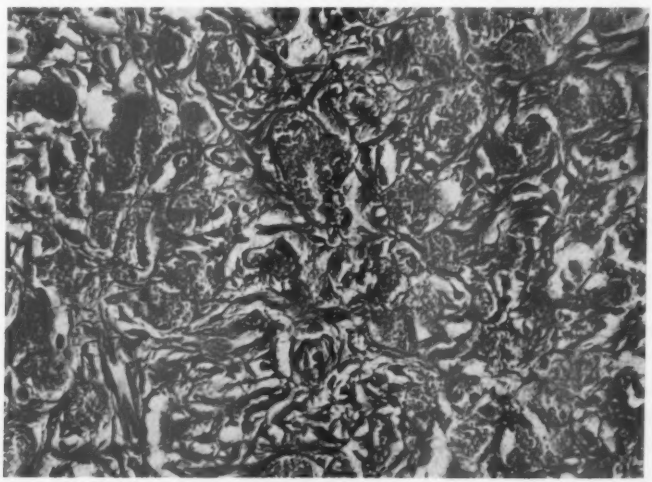

Fig. 4.—Typical polymorphic granular cells of the congenital myoblastoma. The stromal fibrous component is notably greater than that seen in the lingual myoblastomas. (Hematoxylin and eosin stain. Magnification, ×300.)

of the tumor. The tumor cells were polymorphic, ranging from a definite polyhedral to a bizarre spindle form. Reticulin fibers were in intimate association with the cells and, in fact, appeared at times to be a part of the cell membrane (Fig. 4). Staining with Mallory's phosphotungstic acid-hematoxylin and Sudan black B gave the same results as those noted in myoblastomas situated in the tongue.

Histochemical Properties.—The granular cytoplasm was of special interest, since it was thought that it might hold the answer to the origin of the myoblastoma cells. To study better the granules and the association of the tumor cells with the surrounding tissue, the following special staining techniques were employed:

Heidenhain iron-hematoxylin stain. This stain was employed primarily to study the grouping and configuration of the granules themselves. The depth of staining was determined by the aggregate size of the granules, some appearing as fine "dustlike" particles while others were a coarse conglomerate of fine granules. The conglomerate granules were composed of circumscribed aggregates surrounded by a clear zone of cytoplasm.

Mallory's phosphotungstic acid-hematoxylin stain. The granules stained faintly red, while the collagenous elements were red-brown. A poorly vascularized, sparsely cellular collagenous stroma was one of the more pronounced features seen in all cases. Some of the larger collagen bundles ended in a fine reticular pattern which gave them a frayed-out appearance. These reticulin fibers at times blended with the tumor-cell membranes, but the tumor cells did not contribute to the stroma.

Sudan black B stain for fat. This stain was used as a specific for lipids. A faintly positive histochemical reaction was found in the granules of all the lesions, indicating a degree of lipid saturation. The nature and source of these lipids could not be determined.

Periodic acid-Schiff reaction. The granular cytoplasm stained deep red, indicating a possible polysaccharide component. The tissue also was subjected to diastase digestion and then stained with the periodic acid-Schiff reagent. This procedure did not alter the staining properties of the granules, a fact which precluded the presence of free glycogen. Whether this reaction demonstrates a complex molecular form of a glycoprotein could not be determined with any degree of certainty.

Weil's method for myelin sheaths. The cytoplasmic granules stained poorly by this method. Evidence of altered myelin was not demonstrated in the myelin sheaths of the nerves that were included in the sections of tissue. No similarity between the staining characteristics of the cytoplasmic granules of the tumor cells and the myelin of normal nerve could be demonstrated.

Bodian's method of silver impregnation of nerve fibers and nerve endings. Scattered myelinated nerve fibers were seen coursing through the substance of the tumor and, in the majority of cases, were surrounded by a fascicle of tumor cells. There was no evidence of an increase in the number of nerves within the tumors; nor did the configuration of the nerve fibers suggest any structure indicative of specialized nerve endings.

Treatment and Follow-up Data.—Two of the seventeen patients were known to be dead of causes not related to the myoblastoma. One of these patients died thirty years after local excision of the tumor. The second patient died after a radical operation on the glands of the neck which had been performed under the mistaken impression that the lesions in the tongue represented an active squamous-cell carcinoma. Necropsy revealed that the lesions were confined to the tongue. Letters of inquiry were sent to the remaining fifteen patients. Twelve of these queries were answered (eleven in Table I); in no instance was a recurrence reported. In this group of twelve patients the lapse of time between treatment and this study was one to thirty years, the average being 8.5 years (Table I).

COMMENT

Histogenesis.—With the ever-increasing number of reported cases of this tumor, there has been a concomitant expansion of hypotheses as to the origin of the lesions. Four tissues or cell types, or their less-differentiated ancestors, have been proposed as the progenitors of this entity. These are (1) striated muscle, (2) histiocytes, (3) fibroblasts, and (4) neural elements.

Striated muscle: Although several authors[1, 7, 15, 16, 24] accept the idea that the precursor cell originates from striated muscle, there is still conjecture as to whether a degenerative-regenerative process is involved or whether a neoplastic lesion is the prime factor.

On the basis of our investigation and the reports of others,[3, 4, 10, 19] the following observations on the theory of the myogenic origin of the lesion have not been explained:

1. Myoblasts, as seen at any stage of development in the embryo, do not resemble the granular cells of this entity.
2. There is a complete absence of striations in the tumor cells, with no apparent attempts to form them.
3. The transitions between striated muscle and tumor cells have not been demonstrated conclusively, and in our experience the interdigitating striated muscle and tumor cells are separated by an intact sarcolemma. Special staining techniques have failed, in our experience, to link the cytoplasmic granules with the sarcoplasm of striated muscle.

Histiocytes: In 1939 Leroux and Delarue[19] suggested that the cells were mesenchymal in origin, that they were of the histiocytic cell type, and that they were situated in various locations in response to some local stimulus. This concept best explains the variable sites of growth because of the ubiquitous nature

of the histiocyte. Recognizable transitional stages from histiocytes have not been observed; nor have our observations suggested any specificity of substance that might be attributed to phagocytized materials.

Fibroblasts: Pearse,[26] during an intensive histochemical study of these tumors, found that lipids were definitely present, and, on the basis of comparative studies, he concluded that these cells were fibroblasts that had undergone a granular degenerative change.

Our studies revealed a moderate amount of fine granularity in the cytoplasm of the fibroblasts, but this amount by no means was comparable to that seen in the vast cytoplasm of the tumor cells. Projections from the ends of the fibroblasts could be seen to intermingle with the adjacent tumor-cell membranes, but transitional forms as cited by Pearse were not observed.

Neural elements: Recently several authors[3, 4, 10] have presented the theory of the neural origin of these tumors. The nerves encountered in our cases were considered to be the intrinsic nerves of the tongue. In only one instance were tumor cells found within the substance of a nerve. The specialized staining methods did not demonstrate any perceptible changes in the nerves; nor was there evidence that the granularity of the tumor cells was derived from myelin. Schwann-cell proliferation was not seen in any of the many microscopic sections examined. The mere observation that tumor cells are intimately associated with or incorporated within small nerves does not, in our opinion, contribute substantially to evaluation of the origin of this entity.

The origin of these tumors is still an enigma. Our observations, although they partly confirm those of others, do not, in our opinion, lend support to any of the four theories of cell origin; nor have they lent credence to a theory of neoplasia versus reactive tissue elements.

Malignancy.—In passing, it is worth while to reiterate the warning that the epithelial hyperplasia commonly encountered with this entity whenever it is associated with skin or a mucous membrane has never been proved to exhibit malignancy.

An explanation for the hyperplastic nature of the overlying epithelium is not forthcoming. To state that this is a response to the growth of the underlying granular cells is to deny the cases in which there is no apparent epithelial proliferation although the basement membrane is in contact with the tumor cells.

The malignant myoblastoma is a rare and disputed entity. Recently Gamboa[11] reported a case of malignant granular-cell myoblastoma and, after careful review, accepted only ten of the thirty-two cases reported in the literature. In none of these ten cases were the lesions located in the oral cavity, although other authors[6, 15, 20, 22, 28] have reported occurrence of the neoplasms in this site. All the lesions in our seventeen cases were benign and of the uniform cell type. We were impressed by the invasive nature of the tumors in our series; however, the lack of aggressive growth and the favorable response to local treatment confirm the benign cytologic appearance of the tumor cells. On the basis of our experience and that of others, it is questionable whether the benign oral tumors are capable of malignant transformation.

Congenital Myoblastoma.—Some authors consider the congenital granular-cell myoblastoma and the congenital epulis to be separate entities. This distinction is made largely on the clinical basis of age (infants), sex (female), and location (midline of the maxilla).

Custer and Fust,[8] in 1952, observed epithelial inclusions in three of their seven cases. This feature led them to the conclusion that this congenital lesion must be an embryonal hamartoma of a tooth bud rather than a neoplasm. Because of the histochemical similarity of the congenital epulis and the lingual myoblastoma, and because we were unable to identify any epithelial elements suggestive of the primitive tooth structures, we prefer to regard our single case of this particular tumor in a newborn child as a congenital myoblastoma.

Treatment.—Since the granular-cell myoblastoma of the oral cavity is a benign entity, treatment necessarily should be conservative. Any circumscribed lesion on the lateral borders of the tongue, unless it is very extensive, lends itself to excision and biopsy which, in the case of the myoblastoma, have proved sufficient. Roentgen therapy has proved ineffective, since it has been demonstrated that the tumor cells are resistant to roentgen rays.

If, for any reason, routine staining with hematoxylin and eosin is inadequate for true delineation of the cellular components, it is suggested that Mallory's phosphotungstic acid-hematoxylin and periodic acid-Schiff stains be used, since they tend to enhance the morphologic and cytologic clarity of the granular tumor cells.

SUMMARY AND CONCLUSIONS

The results and observations derived from a clinical and histochemical study of seventeen cases of histologically confirmed benign oral granular-cell myoblastoma have been presented.

1. Sixteen of the granular-cell myoblastomas were seen in patients 19 to 62 years old, the average age being 39.9 years. One tumor was observed to arise from the mandibular alveolar ridge of a newborn girl. No particular sex predominance was seen. The usual growth of the lesions was slow and asymptomatic, and they were noted as small, circumscribed, firm nodules on the surface of the tongue, with the lateral border of that structure being the favored site. In one case multiple (three) lesions were seen.

2. Histologically, the myoblastoma was found to be composed of large polygonal cells which contained a characteristic granular cytoplasm. The tumor cells interdigitated with the surrounding fibrous stroma and the adjacent striated muscle, but transitional forms of striated muscle cells or fibroblasts were not found.

3. Peripheral nerves were evident in the sixteen lingual cases of oral myoblastoma, but in only one instance were the tumor cells seen within a nerve sheath. Histochemical studies revealed no evidence of the origin of the tumor cells, although the Sudan black B stain for lipid and the periodic acid-Schiff reaction for a glycoprotein gave positive results in all seventeen cases.

4. In thirteen of the sixteen cases of lingual myoblastoma the overlying epithelium was seen to be hyperplastic and invasive. This feature had been responsible for an original erroneous diagnosis of squamous-cell carcinoma in five of the cases.

5. The single congenital myoblastoma of the alveolar ridge of the mandible gave the same staining reaction as the lingual myoblastoma in adult persons. Microscopic study of several sections of the congenital lesion failed to reveal epithelial structures.

Conservative surgical excision is the treatment of choice.

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ENDODONTICS

The American Association of Endodontists

Captain Warren J. Hedman, Editor

STILL MORE AIDS TO ENDODONTIC PRACTICE

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In two previous articles[1, 2] I listed a number of aids to endodontic practice. In the course of practice I have recorded, from time to time, additional aids which I hope will be of assistance to the dentist in the course of endodontic practice. It is hoped that the aids listed here will help to *keep* one out of difficult situations rather than to *pull* him out.

As before, "while most of the suggestions listed are original with me, some have been included which are not. The latter have been acquired over a period of many years and cannot be traced to their sources."

1. Use thick rubber dam when bleaching a tooth. It will grip the tooth more firmly than thin rubber dam and will prevent leakage of the bleaching agent, which is highly irritating to the gingiva.

2. When about to apply the rubber dam, inform the patient of the reason for its use in order to allay apprehension. If the patient knows that it is being used to keep small instruments from falling down his throat and to prevent irritating solutions from running down into his mouth, he will co-operate much better—and he will not gag.

3. Endodontic treatment is a delicate operation, and instrumentation of the canal should be a precise operation if the instrument is not to be run through the apical foramen and allowed to enter the periapical tissue. Mechanical devices to prevent this are available. A simple, homemade device consists of thick rubber dam cut into squares of about 2 mm., or pieces of wide rubber bands cut to about the same size, and attached to the shafts of all root canal instruments. When in use, the rubber marker is simply slid along the shaft to the correct length of the tooth and the sterilized instrument is carried into the root canal until the marker is level with the incisal or occlusal surface of the tooth.

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4. To control periapical exudate persistently draining into a sterile root canal, immediately before filling the canal irrigate it with hydrogen peroxide solution (3 per cent), dry thoroughly, and fill the canal without delay.

5. It is surprising at times how much debris, formed from inspissated exudate, can be removed with a reamer or file just before the canal is filled. In a narrow canal, this debris is just enough to prevent a gutta-percha or silver cone from reaching to the apex.

6. Root canal instruments must be cleaned of debris and blood before being sterilized. To clean reamers and files, thrust the instrument into the center of a cotton roll so that the instrument is parallel to the length of the cotton roll. Apply slight pressure with the thumb and fingers and remove the instrument by "unscrewing" it or pulling it out counterclockwise. To clean a barbed broach, take a strip of rubber dam about 4 by 2 inches and fold it lengthwise. Insert the broach between the folds of rubber dam, exert slight pressure between thumb and fingers, and withdraw the instrument.

7. To keep root canal cement powder sterile and to dispense it easily, enlarge the hole of a plastic dispenser bottle, preferably one with a spout. Make a small funnel of typewriter paper and sterilize it by means of dry heat at 320° F. for one hour. Do not sterilize the cement powder in this manner, as the heat will cause the powder to fuse into a lump. Simply keep the powder clean and uncontaminated or sterilize with ethylene oxide if this is available. With the help of the funnel, fill the plastic bottle with the root canal cement powder. When the powder is needed, discharge the desired amount and discard that which is left over after mixing the cement.

8. After fitting a gutta-percha or silver cone in the canal, place it in a Dappen dish containing hydrogen peroxide. You may be surprised at the bubbles that are given off while the cone is being cleansed of organic debris or blood serum, as oxygen is being released from the hydrogen peroxide.

9. In placing the outer seal in a posterior tooth, after inserting the cement, apply a strip of rubber dam (about 1 by 3 inches) over the cement. Cover with a cotton roll and apply pressure with the finger or with a ball burnisher. Remove the rubber dam in two or three minutes. This will eliminate an excess of cement but, more important, it will ensure a tight seal, as the cement will set at the margins under pressure.

10. If there is a refrigerator in the office, or a water cooler with a refrigerator compartment, fill empty anesthetic Carpules with water and let them freeze. To carry out the thermal test, simply hold a Carpule in the hand for a moment, press the rubber plunger with an instrument handle to extrude the pencil of ice, and apply the ice to the tooth. It is an excellent test, but do not depend upon it alone to tell you whether the pulp is vital or dead. Used in conjuction with other tests, such as the electric pulp test, it is often significant.

11. Is there gingival bleeding which messes up the undersurface of the rubber dam? Apply a cotton pellet moistened with a small amount of 30 per cent solution of hydrogen peroxide (Superoxol) to the gingiva, and the hemorrhage will stop at once.

12. No canal is really clean of debris unless it has been adequately irrigated. While sterile saline solution may be used as an irrigant, there are advantages in using hydrogen peroxide (3 per cent) and sodium hypochlorite (5 per cent). On interacting, these solutions liberate nascent oxygen which produces an effervescence and thus helps to wash the debris out of the canal. In addition, sodium hypochlorite is one of the best solvents of pulp tissue and organic debris. About 0.5 ml. of each solution is used alternately for irrigating the canal, the needle of the syringe being carried only part of the way up into the canal so as to fit the canal loosely and thus provide an exit through which the solution can drain out of the canal.

13. In irrigating a root canal, two syringes should be used. Since both hydrogen peroxide and sodium hypochlorite are clear, colorless solutions, some means of identifying each solution should be used. This is easily done if one remembers that chlorine is a greenish yellow gas and uses a green Dappen dish exclusively for the sodium hypochlorite. Also, a short piece of green plastic tubing may be placed on the hub of the needle of the syringe which is used for the sodium hypochlorite solution in order to identify it. Or a green-plunger syringe, or a Luer-Lok syringe may be used for the hypochlorite solution and a plain-tip syringe for the hydrogen peroxide.

14. Fracture of a crown just under the gingiva is not a reason for removing the tooth or for operating without a rubber dam. In such cases, the dentist should perform a gingivectomy, incising the gingiva 1 or 2 mm. beyond the line of fracture. This will allow the immediate application of a rubber-dam clamp and execution of the usual endodontic treatment. A periodontal pack need not be applied to the gingiva upon removal of the rubber dam following treatment, although it is a nicety which will take only an additional two or three minutes and is worth the trouble.

15. In narrow root canals it is not always possible to coat the canal wall with cement because the usual gutta-percha plugger cannot be introduced into the canal. In such cases the cement may be introduced into the canal on a reamer or file, which is narrower than the canal, coating the canal wall with a to-and-fro motion of the instrument. On removal, the instrument should be unscrewed or turned counterclockwise so as not to remove the major portion of the cement from the canal.

16. Do not hesitate to tell a patient that a tooth should be treated endodontically. By the same token, do not hesitate to inform a patient that treatment cannot or should not be attempted because of a badly decayed or broken-down crown, severe periodontal condition, nonstrategic or nonfunctioning tooth, etc.

17. If the root canal instrument appears to fit rather tightly, go back to the next-size-smaller instrument, enlarge the canal with it until it fits quite loosely, and return to the next-size-larger instrument. Or cut off 1 mm. of the tip of the instrument which was last used in reaching the apex, change the position of the rubber or metal marker on the instrument to compensate for length, enlarge the canal until the instrument reaches the apex, and then repeat the

procedure of cutting off 1 mm. from the tip of the instrument and using it in the canal. By this time, the canal will have been enlarged sufficiently to take the next size of instrument.

18. If leakage is anticipated after the rubber dam clamp has been applied and the rubber dam is already in place, tie a ligature around the tooth which is clamped. This is done by passing the ligature over the arch of the clamp so as to bring it around the distal aspect of the tooth. Then the lingual end of the ligature is brought forward and is passed through the mesial interproximal space, so that the two ends may be tied buccally.

19. To prevent bending of absorbent points in the hot salt or glass bead sterilizer, pick up at least a half-dozen absorbent points at one time between the beaks of cotton pliers and insert them in the sterilizer, butt end first. This is another practical application of strength in numbers as well as a demonstration of economy of time, as several of the absorbent points will be sterilized simultaneously.

20. In cases where the crown of a tooth is badly broken down or there is subgingival decay, a copper band should be cemented around the crown so as to prevent leakage of the medication or breaking-away of the cement seal. A core of gutta-percha should first be placed to prevent cement from flowing into the root canals. After the band has been fitted, press a thin sheet of wax over the occlusal part of the band and break away the excess wax at the edges. The band has now been converted into a "crown." Fill the band with cement, carry it into place over the tooth, and press it into place by forcing it down with an instrument along the edges until it reaches its destination. This will automatically displace the wax, which should be removed. Add cement occlusally, if necessary. As the cement begins to set slightly, it is carried into the interstices and is molded to shape with an instrument.

21. It is difficult at times to locate a root canal orifice in a molar tooth, especially if the orifice is very narrow and is filled with debris. In such cases, a broken No. 17 explorer, the tip of which has been sharpened to a fine point, will help one to find, and to break into, the orifice of the root canal.

22. To identify the sizes of root canal instruments quickly, use nail polish of different colors to paint the handles of the instruments (for example, dark red for No. 10, medium red for No. 15, light red for No. 20, dark orange for No. 25, light orange for No. 30, yellow for No. 35, and so forth, repeating the colors serially for the remainder of the instruments). In other words, use the graded colors of the rainbow, as available, to differentiate the sizes of instruments.

23. Patients will be more comfortable if a topical anesthetic is sprayed on the gingiva of the tooth to be treated prior to the application of the rubber-dam clamp. This will not only prevent the patient from feeling the pressure of the clamp, but it will also facilitate slipping the rubber dam over the tooth.

24. For preliminary drying of the root canal, convert the irrigating syringe into an aspirator by inserting the needle into the root canal and pulling back

on the plunger to create suction, or else simply place the hub of a Luer-Lok type of needle in the rubber tip of a saliva-ejector holder and thus convert it into an aspirating tip.

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4001 SPRUCE ST.

ENDODONTIC TREATMENT OF AN UPPER FIRST MOLAR IN A HEMOPHILIAC

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CASE REPORT

A 9-year-old boy was referred to the College of Dentistry for endodontic treatment of the upper left first molar. At the time, the boy was a patient in the State University of Iowa Children's Hospital. He and three of his brothers were true hemophiliacs and were frequently in the hospital with minor cuts and bruises which always required hospitalization and numerous blood transfusions. The patient and two of his brothers had severe hemophilia. This time he had been in the hospital for eight days, following a small cut in the scalp above his right eye. The treatment had required eleven pints of blood or plasma.

Fig. 1.

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Local Findings.—The left upper first molar was slightly sensitive to hot and cold and tender to percussion following a pulp-capping procedure. The patient had complained of discomfort ever since the pulp capping was placed. Roentgenographic examination showed no periapical involvement and no proximal caries. There was no response to the electric pulp tester. No swelling or discomfort was evident on the buccal aspect of the alveolar process.

Treatment.—A local anesthetic was given, the rubber-dam was applied in the usual way, and the tooth was entered through the occlusal surface. The coronal pulp was found to be necrotic. No bleeding was experienced before the root canals were entered. Removal of the root pulps with barbed broaches started profuse bleeding immediately. The root canals were enlarged with reamers and files and irrigated with 3 per cent hydrogen peroxide. The bleeding did not stop, however, until the root canals were filled with small wisps of cotton soaked in 1 per cent epinephrine hydrochloride. When the bleeding had subsided, the pulp chamber was thoroughly wiped out with camphorated paramonochlorophenol, the cotton soaked in epinephrine was removed, and a gentle stream of warm air was used to dry the canals and pulp chamber. When dry, the tooth was filled with sealer and silver points (Fig. 1). A cement base and an occlusal filling were inserted immediately.

Fig. 2.

Results.—There was no discomfort immediately after the treatment, and the tooth has been completely asymptomatic ever since. No periapical area was visible after nine months of observation (Fig. 2), and there is no soreness or tenderness to percussion.

DISCUSSION AND CONCLUSION

In the case just presented, endodontic treatment provided a comparatively easy solution to a difficult problem. Had it been necessary to extract the tooth, it would have been a major surgical procedure requiring from five to ten pints of blood or plasma, and the patient would have been hospitalized for not less than one week. The root canal filling was not perfect, but it was imperative that it be done quickly and at one visit. The outlook for saving the tooth appears to be good.

RESEARCH

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MITOTIC ACTIVITY OF HUMAN ORAL EPITHELIUM EXPOSED TO 30 PER CENT HYDROGEN PEROXIDE

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INTRODUCTION

ACCORDING to the findings reported by Meyer[1] and her associates, the frequency of mitosis in epithelium of human gingiva is 50 per cent higher in older age groups (50 to 78 years) than in younger age groups (25 to 35 years). This serves to support Bullough's[2] findings of considerably higher mitotic activity in the epidermis of middle-aged mice as contrasted with young animals. These findings contradict previously accepted concepts of aging. It is generally accepted that bodily processes slow down and the regenerative capacity of the tissues diminishes with age. Marwah and Weinmann[1] state that one might relate carcinogenesis in older age groups to the possibility of loss of the regulative mechanisms governing mitotic frequency and normal cellular proliferation.

Orban,[3] investigating the effects of a 30 per cent hydrogen peroxide solution on chronically inflamed human gingiva, incidentally reported an apparent simultaneous increase in the mitotic activity of the epithelium. This additional finding established the purpose of the study reported here.

We made this study in order to investigate, in two age groups, the morphologic changes of the normal gingival epithelium when influenced by this chemical stimulant, and thus to establish more quantitatively valid data.

The findings will be significant in establishing norms of mitotic index, mitosis patterns, cell density, and their possible alterations under the direct influence of a cellular stimulant, especially in the older persons.

REVIEW OF LITERATURE

The early investigations of mitotic activity were concerned with describing and labeling the various processes and stages involved.[4, 5] Flemming[6]

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made the first quantitative study of mitotic activity in an attempt to correlate this activity with cell loss and replacement.

In 1908 Minot[7] added further impetus to the quantitative analysis of mitotic activity by studying the epidermis of rabbit embryos at varying age ranges. He found different mitotic indices in various tissues, thus establishing evidence of differential rates of growth. Minot described the mitotic index (MI) as "the number of cells to be found at any given moment in the active process of division out of a total of 1,000 cells." This index has been a very useful tool for many years.

Variations in Mitotic Activity.—

Regional differences: Thuringer[8, 9] established the earliest quantitative counts of cell division. He counted one mitosis for 2,414 cells on the scalp, for 378,325 cells on the leg, and for 268,275 cells on the ear of a human subject.

In 1938 Cooper and Schiff[10] reported a mitotic index of 4.6 in an 8-day-old human prepuce.

In 1953 Pinkus[11] used Minot's mitotic index to chart many past findings. In his review it is seen that Thuringer's mitotic index of adult human epidermis ranges from 1.6 to 0.1. A summary of these values is found in Table I.

TABLE I. MITOTIC INDEX (HUMAN EPIDERMIS)

AUTHOR AND YEAR	REGION	AGE	MITOTIC INDEX
Thuringer (1924)	Scalp	Adult	10 mitoses per sq. mm.
Thuringer (1928)	Prepuce, scalp, ear, leg	3 years	0.74
		Adult	0.41
		Adult	0.0037
		Adult	0.0026
Cooper (1939)	Prepuce	Newborn	1.4-6.8 (Average, 3.8)
Cooper (1939)	Prepuce	6-11 days	4.1
Broders and Dublin (1939)	Prepuce	Newborn	7.4
Andrew and Andrew (1949)	Antecubital region	Adult	0.13
Katzberg (1952)	Abdominal skin	0-20 years	Basal 0.23 Spinous 0.26
		21-40 years	Basal 0.48 Spinous 0.34
		41-60 years	Basal 0.73 Spinous 0.37
		61-80 years	Basal 0.76 Spinous 0.36
Pinkus (1952)	Forearm, flexor surface	46 years	1.59

Mitotic rhythmicity: It is generally recognized that a periodic mitotic rhythm exists in the regeneration of plants and animals. Rhythmic cycles in plants were first established by Killicott[12] in 1904. Fortuyn-Van Leyden,[13] in 1916, was the first to report this phenomenon in animals.

Many investigators have shown the existence of a daily rhythm in both animals and man. As yet, however, there is no agreement as to the timing of peak mitotic activity.

Henry[14] found a maximal activity of 7.2 and a minimal activity of 3.8 mitoses per 1,000 cells in the oral mucosa of a 3½-month-old rabbit at 1 P.M. and 10 P.M., respectively.

Halberg and associates,[15] in 1952, reported a night low and a morning high in mitoses in the retromolar epithelium and periodontal membrane of 5-month-

old black male rats. No day-and-night difference was detected in the interdental papilla.

These discrepancies may be due to differences in species, methodology, laboratory conditions, or biometric analysis.

Distribution of mitosis: The site of regenerative activity in the epithelium is the stratum germinativum.

Thuringer,[8] Henry,[14] and Marwah[1] have gathered data on the distribution of mitosis in the different strata. Thuringer found 12 per cent of mitoses in the basal layer and 88 per cent in the spinous layer of cat epidermis. Henry found 60 per cent in the basal layer and 40 per cent in the spinous layer of rabbit buccal mucosa. Marwah reported 23 per cent of mitoses in the basal layer and 77 per cent in the inner spinous layer of human attached gingiva. These values are summarized in Table II.

TABLE II. DISTRIBUTION OF MITOSIS

AUTHOR AND YEAR	SOURCE OF MATERIAL	REGION	DISTRIBUTION OF MITOSIS	
			BASAL LAYER (PER CENT)	SPINOUS LAYER (PER CENT)
Thuringer (1924)	Adult man	Scalp	12	88
Thuringer (1939)	Cat	Foot pad	44.5	55.5
Cowdry and Thompson (1944)	Mouse	Foot pad	27.5	72.5
Thuringer and Cooper (1950)	Adult man	Abdomen	-50	+50
Pinkus (1952)	Adult man	Forearm	61	39
Henry (1952)	Rabbits	Oral buccal mucosa	60	40
Marwah (1956)	Adult man	Attached gingival epithelium	(25 yr.) 22 (50 yr.) 24	(25 yr.) 78 (50 yr.) 76

Some earlier investigators, prompted by the supposedly insufficient rate of epidermal mitoses, attributed regeneration to the underlying connective tissue[16, 17] and the transformation of lymphocytes into epithelial cells.[18]

TABLE III. MITOTIC INDEX—ORAL MUCOSA (MALE)

AUTHOR AND YEAR	SOURCE OF MATERIAL	REGION	AGE	MITOTIC INDEX
Henry (1951)	Rabbits	Buccal mucosa	3.5 mo.	5.1
Halberg (1954)	Rats	Retromolar epithelium	5 mo.	11.0
Muhlemann (1954)	Rats	Retromolar epithelium	5 mo.	4.4
Muhlemann (1955)	Rats	Interdental epithelium and epithelial attachment	5 mo.	39.81
Marwah (1956)	Human	Attached gingival epithelium	25-35 yr.	0.98
			50-78 yr.	1.56

Mitotic Activity in Oral Epithelium.—The first mitotic index of human oral mucous membrane was published by Marwah[1] in 1956. He reported the age difference in the mitotic index of the human attached gingival epithelium. The ages ranged from 25 to 35 years and from 50 to 78 years. The older group was found to have 50 per cent more mitotic activity than the younger

group; thus, it is seen that gingival epithelium behaves like the epidermis of man and mice. Marwah reported an average mitotic index of 0.98 in the young group and 1.56 in the older group.

Mitotic indices have been determined in the buccal mucosae of adult rabbits by Henry[14] and in several areas of the adult rat gingiva by Halberg[15] and Muhlemann.[19] Henry's main objective was to establish the pattern of mitotic rhythmicity in the oral mucosa of rabbits. These values are summarized in Table III.

Factors Influencing Mitotic Activity.—Among the factors which will effect the daily variation in mitotic activity are the following:

1. *Physical stimulation.* Carleton[20] found that continuous exposure of animals to light caused an alteration in the rhythmical periodicity of mitosis.

2. *Hormonal.* Bullough[21] presented evidence that the estrone levels in the female mouse effected the renewal of epidermal cells.

3. *Temperature.* Bullough[2] concluded that mitotic activity is high during lowered temperatures and low during higher temperatures.

4. *Blood sugar levels.* Bullough[2] observed the existence of a direct relationship between mitotic activity and blood sugar levels.

5. *Glucose oxidation.* Bullough[22] concluded that mitotic activity could be increased by stimulating the glucose oxidation. In order for this action to be effective, it had to occur just prior to prophase.

6. *Stress.* Bullough[23] demonstrated that stress is an influencing factor in mitotic activity. The glucocorticoid hormones play a role in the antimitotic mechanisms, while testosterone was seen to induce mitotic activity.

7. *Age.* Marwah[1] reported a marked increase in mitoses in older human gingival epithelium. On the other hand, Kiljunen[24] reported the mitotic index in the epidermis of young rats to be higher than that of older ones.

Action of Oxygen on Epithelial Tissues.—Rothman[25] believes that there is evidence that the main steps in energy metabolism in the skin proceed as in other tissues, namely, glycolysis of carbohydrates, oxidation of the products of glycolysis, fats, and amino acids via the Krebs citric acid cycle, and finally a transfer of electrons through the cytochrome system to molecular oxygen.

The cellular portion of epithelium, exclusive of the inert stratum corneum, is more active than other tissues in the utilization of oxygen.

Orban,[3] using a repeated application of 30 per cent hydrogen peroxide which in turn produced monomeric oxygen, was able to produce significant changes on human gingival epithelium. The basal-cell layer was found to form from four to six layers of cells with a remarkable increase in mitoses. He attributed an epithelial proliferation and an intensified hornification to the great increase in mitotic activity. In addition, there was a marked decrease in the inflammatory cell infiltration.

In 1949 Bullough[26] reported the relationship between the epidermal mitotic activity and the blood sugar level of the mouse. He found that epidermal glycogen content increases during sleep when the mitotic rate increases. With the onset of sleep, glucose is deposited in the tissue in the form of glycogen.

Bullough considers the glucose, or glycogen, to be the critical substance effecting the mitotic activity in the epidermis of the adult mouse and of man.

Glickman and colleagues[27] discouraged the assumption that the artificial introduction of oxygen would appreciably hasten the normal cellular processes in the course of gingival healing.

Bullough[22] investigated the relationship of mitotic activity and oxygen tension. The number of mitoses can be increased by stimulating the rate of energy production from glucose oxidation. This has its effect immediately prior to the prophase (antephase) which appears to be a critical time in the histophysiology of mitosis.

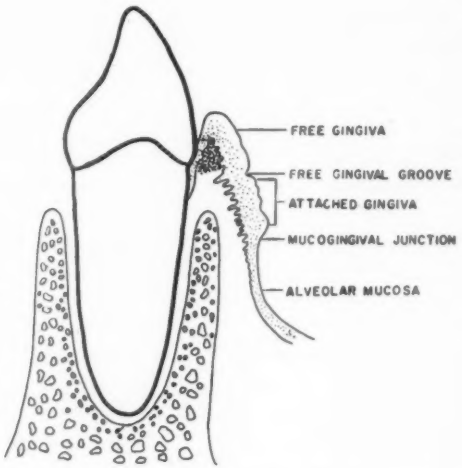

Fig. 1.—Diagrammatic illustration of the oral mucous membrane in the region of the dentogingival junction.

Bullough[23] reported mitotic activity as being related to some stage of carbohydrate metabolism. Thus, carbohydrate supply and utilization (oxidation) are the relative important factors. Tissues that have a daily mitotic cycle (for example, epithelium) are at the lower end of a priority scale in glucose supply and utilization. Thus, these tissues are normally "half-starved" in spite of being constantly profused with the glucose of the blood stream.

Holman[28] feels that agents which inactivate catalase or produce hydrogen peroxide should have a detrimental effect on tumors.

MATERIAL AND METHODS

The area selected for a study of the mitotic activity of normal oral epithelium, prior to and after treatment with a 30 per cent hydrogen peroxide solution, was the attached interdental gingival epithelium (Fig. 1). This portion of the masticatory oral mucosa was used because of the ease with which it may be

biopsied and because other workers[1] had previously established a mitotic index for this tissue. The investigation of normal interdental gingiva also permitted an independent evaluation of the mitotic index in different age groups.

Only human adult male subjects were used, since the hormonal influence of the menstrual cycle in women could add a variable influencing the rate of mitosis.

The biopsy contributors were thirty healthy men. Fifteen men ranging in age from 22 to 27 years comprised the younger age group, and the other fifteen, ranging in age from 48 to 63 years, made up the older age group.

A biopsy specimen was taken from each subject before the experimental treatment in order to establish the norms of mitotic activity.

Each donor received three applications of a neutralized 30 per cent hydrogen peroxide solution (Superoxol, a product of Merck & Company, Inc.) per week for a period of thirty days. The agent was applied directly to the same interdental papillae by means of a small cotton pellet held in cotton pliers (Fig. 2). This was allowed to remain for one minute and then was irrigated well with plain tap water. This particular time interval was previously established in pilot studies. Previous pilot studies of normal human gingiva were performed in order to determine the best technique of biopsy, the best fixative agent, the best stain technique, the effect of single and multiple applications of the 30 per cent hydrogen peroxide solution, the optimal dosage of the solution, and the best time for biopsy of the oxygenated specimens. The thirty-day interval was selected because of its apparent optimal mitotic activity.

Biopsy Technique and Specimen Preparation.—All the biopsy specimens were taken between 11:30 A.M. and 12:30 P.M. in order to minimize any variation in mitotic counts that might be due to a time factor.

The tissue which was exposed to the 30 per cent hydrogen peroxide was biopsied twenty-four hours after the last application. These specimens will later be referred to as the "oxygenated experimental group," in contrast to the "normal control" group.

In preparation for biopsy, the alveolar mucosa was infiltrated with a local anesthetic agent. The interdental papillae between the lower premolars and molars was excised. The tissue was washed in room-temperature tap water and immediately fixed in Zenker-formalin solution for eight hours. The specimens were then washed in cold tap water for twenty-four hours. The tissue was embedded in paraffin, sectioned at 6 microns, and stained with hematoxylin and eosin. The sections were cut at right angles to the surface of the epithelium.

Method of Analysis.—One section of each specimen was selected at random. Tracings of microscopic projections from each specimen, magnified 100 times, were made. The exact magnification was determined each time by projecting a stage micrometer to verify the measurement. The boundary between the

free and attached interdental gingiva was demarcated. The attached interdental gingiva selected for study was free of inflammatory cells. The attached interdental gingiva was examined microscopically (high dry, ×500) for the presence of epithelial cells in the active process of mitosis. The mitotic figures were located on the magnified tracings and their phase, location, and plane of division were recorded in chart form.

Measurement of Cell Population.—A surface planimeter, calibrated in square centimeters, was used to measure the total surface area of epithelium. A small area of epithelium was designated on the tracing as the sample area.

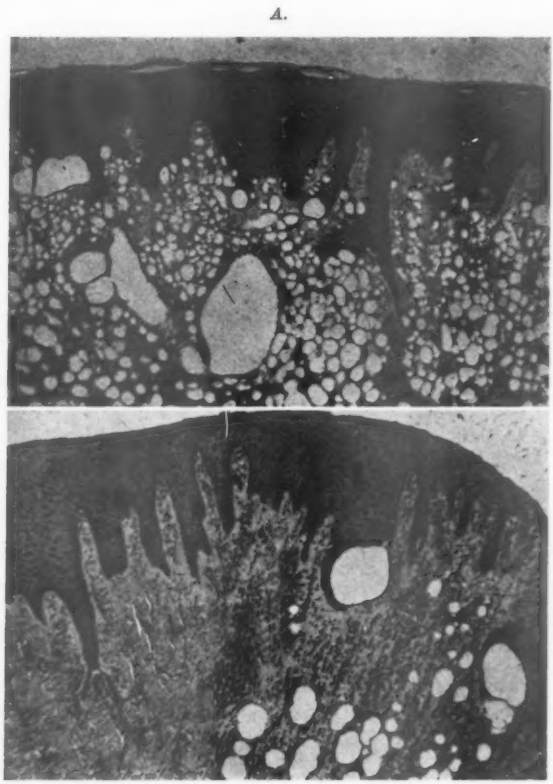

Fig. 2.—*A*, Photomicrograph of attached gingiva fifteen minutes after application of 30 per cent hydrogen peroxide. Notice the many "oxygen bubbles" in the underlying connective tissue.
B, Gingiva thirty minutes after hydrogen peroxide application. Only a few bubbles remain in the connective tissue.

This sample surface was measured, and the total number of cells within its boundaries were counted with the aid of a hand-tally mechanical counter. From this, the total number of epithelial cells in the entire attached gingiva of the specimen was computed.

Mitotic Index.—The mitotic index, expressed as the number of counted mitotic figures per 1,000 cells, was computed as a simple proportion utilizing the estimated total cell population and the total mitotic count.

In addition, each specimen was examined for the degree of hornification, suprapapillary thickness of epithelium, height of the basal cells, and presence or absence of a granular-cell layer. The number of cell layers in the stratum spinosum was also recorded. All measurements were taken with a disc micrometer.

Phases of Mitoses.—The stages of mitoses were identified according to established standards,[29] as follows:

1. *Prophase* was identified by an increase in the size of the cells, an increased visibility of chromatin threads, the cell's tendency to assume a spherical form, and an increased basophilia of the nucleus.
2. *Metaphase* was recognized by the absence of a nuclear membrane and the arrangement of chromosomes in an equatorial plate.
3. *Anaphase* was characterized by a divergence of chromosomes from the equatorial plate in the undivided cell body.
4. *Telophase* was recognized by the constriction or division of the cell body into two daughter cells.

FINDINGS

Mitotic Index.—

Control biopsy specimens: The mitotic index (MI) represents the number of observed mitoses per thousand cells in the total epithelium. The average mitotic index for the younger age group (25 years) was 0.79, and the average for the older age group (56 years) was 1.69. Thus, the average mitotic index was approximately twice as high in the group of older persons.

The difference between the two age groups in the distribution of mitotic indices is further demonstrated in Fig. 3. All the mitotic indices are divided into three large divisions. Those ranging from 0 to 0.5 were found in six of the fifteen specimens from the younger men and one of the fifteen specimens from the group of older subjects. Indices ranging from 0.5 to 1.5 were found in seven of the younger and seven of the older age groups, respectively. An index above 1.5 occurred in two specimens from the younger age group and seven from the older age group. From this, it is seen that the most striking contrast lies in the higher incidence of a mitotic index of from 0 to 0.5 in the younger age group, as opposed to the greater frequency of a mitotic index above 1.5 in the older age group.

Experimental biopsy specimens: After treatment with 30 per cent hydrogen peroxide, the average mitotic index of the fifteen young persons increased to 6.50. In the older age group the index increased to 8.76. The mitotic index in the younger age group had increased from 0.79 to 6.50, or approximately eight times. In the older age group the mitotic index had increased from 1.69 to 8.76, or about five times (Fig. 4). Examples of cells undergoing mitosis and the increased accumulation of mitotic figures in cell nests can be seen in Figs. 5 and 6.

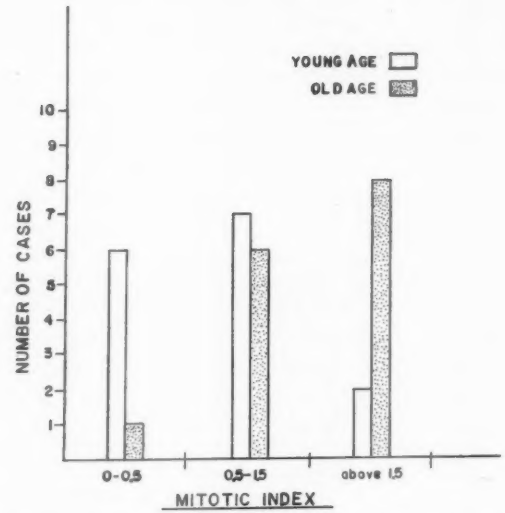

Fig. 3.—The range distribution of mitotic index in both age groups of the control specimens.

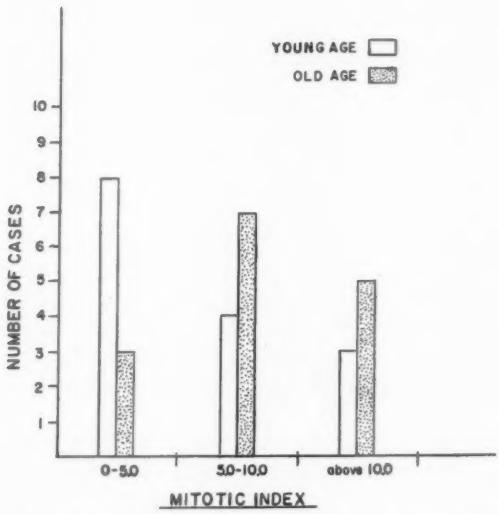

Fig. 4.—The range distribution of mitotic index in both age groups of the experimental specimens.

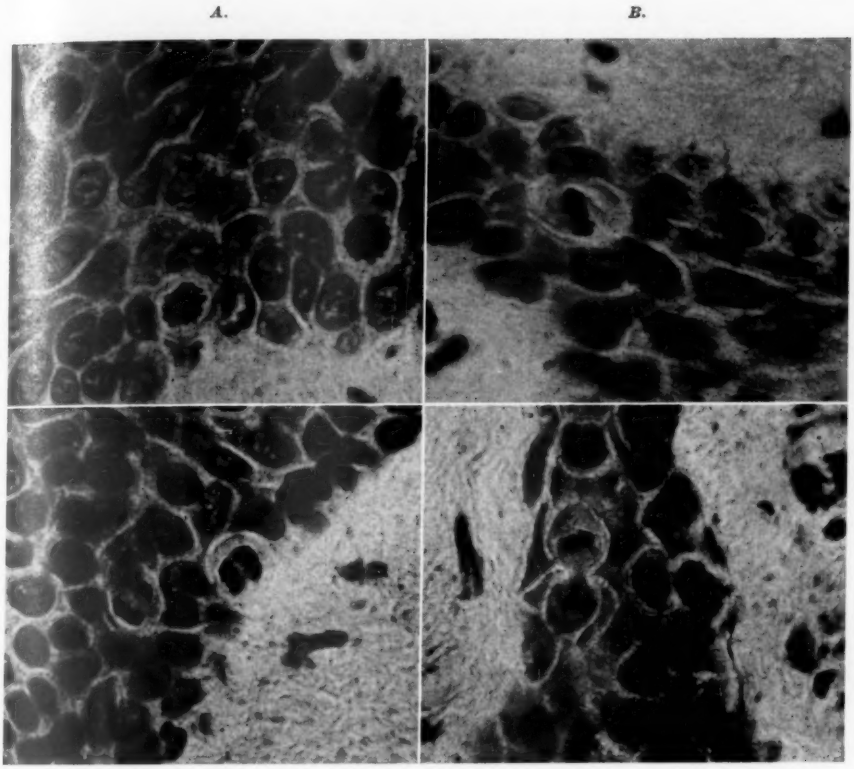

Fig. 5.—Examples of mitotic figures from tissue which was exposed to 30 per cent hydrogen peroxide. *A*, Prophase; *B*, metaphase; *C*, anaphase; *D*, telophase (note the intermediate body of Fleming).

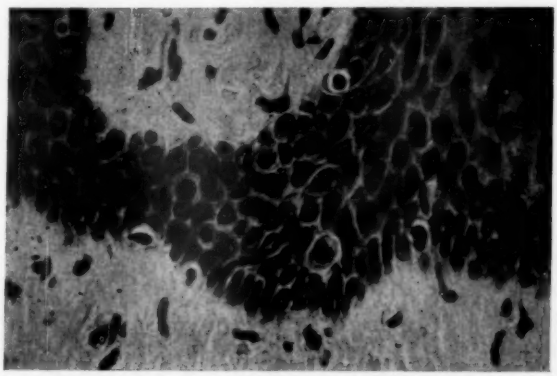

Fig. 6.—Accumulation of mitotic figures in cell nests following prolonged exposure to 30 per cent hydrogen peroxide.

The mitotic index ranged from 1.4 to 22.3 in the younger men and from 4.6 to 15.7 in the older men. Once again the mitotic indices are divided into three large divisions. Those ranging from 0 to 5.0 were found in eight of the fifteen specimens from young men and in three of the fifteen specimens from older men. Indices ranging from 5.0 to 10.0 were found in four specimens from the younger age group and seven of the fifteen specimens from the older age group. An index above 10.0 was found in three of the young men and five of the older men. Comparisons of mitotic index in both age groups and specimens can be seen in Fig. 7.

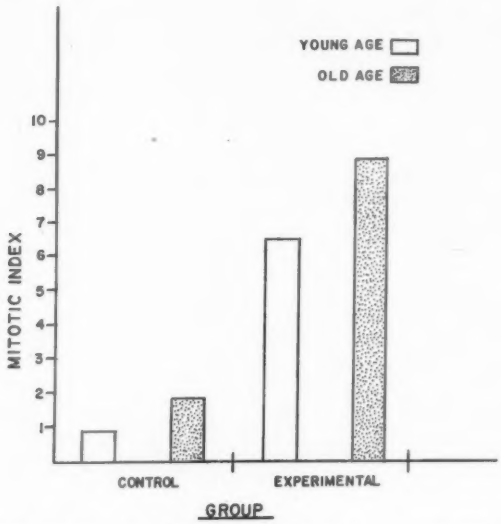

Fig. 7.—A comparison of mitotic index in both age groups and specimens.

Cell Density and Its Relation to Mitotic Index.—

Control biopsy specimens: The cell densities were plotted against age and the mitotic index. The number of cells per $(100\ u)^2$ averaged 62 for the younger age group and 70 for the older age group. There was no direct correlation between cell density and mitotic index.

Experimental biopsy specimens: The number of cells per $(100\ u)^2$ averaged 58 for the younger age group and 65 for the older age group. The over-all cell density in the experimental specimens was seen to decrease.

Differential Counts of Phases in Mitosis.—

Control biopsy specimens: The frequency distribution of the various phases of mitosis in the control specimens is tabulated in Table IV.

The respective equivalent proportion of cells in prophase, metaphase, anaphase, and telophase is as follows:

Younger age	2:3:2:4
Older age	4:–2:2:3

TABLE IV. PER CENT OF MITOTIC PHASES

AGE GROUP	TOTAL NO. OF MITOSES	PROPHASE (PER CENT)	METAPHASE (PER CENT)	ANAPHASE (PER CENT)	TELOPHASE (PER CENT)
		Control Group			
1. Young	50	18	28	18	36
2. Old	150	37	16	18	29
		Experimental Group			
3. Young	439	40	25	11	23
4. Old	456	56	28	5	11

A total comparative proportion of the above percentages would be as follows:

(1) 2: 3: 2: 4
(2) 4:-2: 2: 3
(3) 10:-6: 2: 1
(4) 11: 6: 1: 2

Experimental biopsy specimens: The distribution of the various phases of mitosis in the experimental specimens is tabulated in Table IV.

The respective equivalent proportion of cells in prophase, metaphase, anaphase, and telophase is as follows:

Younger age	10:-6:2.2:1
Older age	11:6:1:2

Location of Cells in Mitosis and Their Respective Planes of Division.—

Control biopsy specimens: Mitosis occurred either in the basal-cell layer or in the deeper layers of the stratum spinosum. Almost one-half of the total number of mitotic figures in either age group were in the basal-cell layers and the remainder were in the stratum spinosum.

An attempt was made to determine the plane of division of mitosis in relation to the basement membrane. Since it is difficult to ascertain the plane of division of cells in prophase and early metaphase, these phases were eliminated from this tabulation.

Sixty-five per cent of the two recorded phases were perpendicular to the basement membrane. The remaining 35 per cent were dividing in a parallel plane. There was no significant difference between the two age groups. These figures are a combined sample for the cells undergoing division in both the basal- and prickle-cell layers (Table V).

TABLE V. LOCATION AND PLANES OF MITOSIS

AGE GROUP	TOTAL NO. OF MITOSES	LOCATION OF MITOSIS		PLANE OF MITOSIS	
		BASAL-CELL LAYER (PER CENT)	PRICKLE-CELL LAYER (PER CENT)	PERPENDICULAR (PER CENT)	PARALLEL (PER CENT)
		Control Group			
Young	50	48	52	61	39
Old	150	51	49	68	32
Average for both groups	200	49.5	50.5	65	35
		Experimental Group			
Young	439	54	46	56	44
Old	456	55	45	69	31
Average for both groups	895	54.5	45.5	62.5	37.5

Experimental biopsy specimens: There was no significant difference in the two age groups. Fifty-five per cent of the total number of mitotic figures observed in both groups were in the basal-cell layer, and 45 per cent were in the prickle-cell layer. Sixty-three per cent were perpendicular, and 37 per cent were parallel to the basement membrane.

Despite the great increase in mitotic index, the ratio of the planes of division is almost the same in the control and experimental groups (Table V).

Degree of Keratinization, Suprapapillary Epithelial Thickness, Height of Basal Layer, and Incidence of Granular Layer.—

Control biopsy specimens: A cornified layer was present in twenty-nine of thirty cases. In different areas of the same specimens the cornified seam ranged in thickness from 3.2 to 17.0 microns. There was some apparent variance in thickness of the keratin layer between the younger and older age groups. The thickness of the keratin layer in the younger age group ranged from 3.2 to 16.0 microns, with an average of 8.16. In contrast to this, the range in older age group was from 6.0 to 17.0 microns, with an average of 14.10 (Table VI). There was no apparent correlation between the degree of cornified layer and the mitotic index.

TABLE VI. MEASUREMENTS OF KERATIN LAYER, SUPRAPAPILLARY EPITHELIUM, AND BASAL-CELL LAYER

AGE GROUP	KERATIN LAYER THICKNESS (MICRONS)		SUPRAPAPILLARY EPITHELIAL THICKNESS				BASAL-CELL LAYER HEIGHT (MICRONS)	
			RANGE CELL LAYERS	AVERAGE CELL LAYERS	RANGE (μ)	AVERAGE (μ)	RANGE (μ)	AVERAGE (μ)
	RANGE	AVERAGE						
Control Group								
Young	0-16	8.16	11-16	12.9	70-125	106	4-9	6.38
Old	6-17	14.10	10-15	13.1	76-160	126.7	6-9	6.81
Experimental Group								
Young	3-19	11.90	9-17	14.3	80-144	124	6-12	10.10
Old	0-25	14.40	10-21	13.1	76-144	121	6-15	10.10

The suprapapillary epithelial width measurements were recorded. This measurement was difficult because of the uncertainty of the tissue specimen's definitely being cut at an absolute right angle to the surface. In order to reduce the errors in this measurement, specimens with a tangential section were eliminated. Three readings were taken for each specimen, and these were averaged. The measurements were taken from the tip of the papillae to the base of the keratin layer. The number of cell layers in the younger age group ranged from 11 to 16, with an average of 12.9 rows. In the older age group these values ranged from 10 to 15 cell layers, with an average of 13.1. The epithelial thickness in the younger age group ranged from 70.4 to 125.6 microns, with an average of 106.0 microns. In the older age group this thickness ranged from 76.8 to 160 microns and averaged 126.7 microns (Table VI).

A final measurement was made of the height in a single row of basal cells. There was no significant difference between the two age groups. In the younger age group the height of the cells varied from 4.0 to 9.6 microns and averaged

6.38 microns. In the older age group the range was from 6.4 to 9.6, with an average of 6.81 microns.

Experimental biopsy specimens: A hornified layer was observed in twenty-nine of the thirty specimens examined. The cornification varied in degree, but it appeared to be somewhat more compact than in the normal specimens. The thickness of the keratin layer in the younger age group ranged from 3.0 to 19.2 microns, with an average of 11.9. In the older age group it ranged from 3.2 to 25.6 microns and averaged 14.4 (Table VI).

The suprapapillary epithelial width was ascertained in the same manner as before. The number of cell layers in the younger age group ranged from 9 to 17, with an average of 14.3 rows. In the older age group these values ranged from 10 to 21, with an average of 13.1 cell rows. The thickness in the younger age group ranged from 80.0 microns to 144.0 microns and averaged 124.4 microns. In the older age group it ranged from 76.8 to 144.0 microns and averaged 121.5 microns (Table VI).

The height of the basal-cell layer once again showed no difference between the two age groups. However, there was a marked contrast between the basal cells of the control and experimental groups. In the experimental specimens the basal-cell layer was more "columnar" in appearance; it was well demarcated and the pedicles were more prominent. In the younger age group the height of the cells ranged from 6.0 to 12.8 microns, with an average of 10.1. In the older age group it ranged from 6.4 to 15.0 and averaged 10.1 microns.

DISCUSSION

Possible Mechanism of Hydrogen Peroxide Action.—The induced increase of the mitotic index following prolonged exposure to 30 per cent hydrogen peroxide appeared in both extremes of age. In an attempt to explain the greatly increased mitotic index, several points must be taken into consideration.

The first point which one might obviously consider is that the increased mitotic index is a true expression of a proportional increase in mitotic activity. The increased mitotic index need not represent an associated increased mitotic activity, however, but possibly a retardation or arrest of mitosis similar to that produced by colchicine. In addition to the two aforementioned points, the increased mitotic index may be due to a relative increase in mitoses and associated with a more prolonged mitotic period in one of the phases.

From the viewpoints which have thus far been expressed, the latter appears to be the more likely phenomenon. This statement is based on two observations which may possibly be the key to the mechanisms involved. The first observation is based upon a clinical finding of the tissue specimen before biopsy. When this tissue was first exposed to the 30 per cent hydrogen peroxide, there was extreme "blanching" for a prolonged period. Following repeated exposure, and at the end of the thirty-day period, the tissue did not blanch easily or as intensely as it did in response to the earlier applications. In addition to this clinical phenomenon, we observed that the mitotic index does not show a dramatic increase until after a lag period of three weeks following the initial application of hydrogen peroxide.

The possible mechanisms to explain the above-mentioned phenomenon may lie in alterations of the normal tissue's metabolic processes. If two hydrogen atoms combine with an oxygen molecule, in normal tissue respiration, the product is not water but hydrogen peroxide. The hydrogen peroxide is a strong oxidizing agent and in any quantity is very poisonous to cells. In accord with this, there are two enzymes (oxidases) which are responsible for the breakdown of the possible toxic accumulation of hydrogen peroxide in animal tissue. These are catalase and peroxidase.

The function of tissue catalase appears to be in the splitting of hydrogen peroxide, which might otherwise accumulate during biologic oxidation and be toxic to living cells. Catalase will aid in the breakdown of hydrogen peroxide to yield gaseous oxygen[30]:

$$2\ H_2O_2 \xrightarrow{\text{Catalase}} 2\ H_2O + O_2 \quad \text{(Molecular oxygen)}$$

Peroxidase, if present, will act upon hydrogen peroxide and organic peroxides to yield an active oxygen which, in turn, oxidizes the substrate. These are usually phenols:

$$H_2O_2 \xrightarrow{\text{Peroxidase}} H_2O + O \quad \text{(Atomic oxygen)}$$
$$M + O \qquad MO \quad \text{(Metabolite)}$$

It is further hypothesized that cells in the area of prolonged hydrogen peroxide exposure have lessened or exhausted their ability to produce sufficient catalase; hence, the clinically observed phenomenon of less "blanching" in the later days of hydrogen peroxide exposure. This may be due to a diminished breakdown of hydrogen peroxide; thus, some unknown quantity of hydrogen peroxide would be able to enter the tissue and produce a possible poisoning of the cells.

An increased mitotic activity, to compensate for the increased loss of the poisoned cells, may well be the explanation for a true proportional increase in mitotic index. On the other hand, a colchicine-like action of the hydrogen peroxide may have occurred with only a relative increase in mitoses and a prolonged period in one of the phases.

The latter viewpoint gains credence when we review the work on the action of hydrogen peroxide in controlling tumorous growths. This phenomenon of retarding cell growth with the use of peroxide has been used experimentally in the treatment of adenocarcinoma.[28] Numerous animals and some human beings have already been treated with marked results.

Holman feels that agents which inactivate catalase or produce hydrogen peroxide should have a detrimental effect on tumors. In essence, this same thing is thought to occur in the use of radiation therapy. Ionizing radiations have long been known to destroy malignant cells, and this may be explained as being due to the production, in the presence of oxygen in fluid systems in vivo, of hydrogen peroxide, the free powerful oxidizing radicals HO_2, HO, and atomic oxygen as well as the destruction of the enzyme catalase. It is a known fact that tumor cells are low in catalase; thus the peroxides are able to enter the tissue and retard their uncontrolled growth.[31]

In various experiments[28, 32, 33] hydrogen peroxide was administered by intraperitoneal injection, by intravenous injection, or simply by placing small quantities of hydrogen peroxide in drinking water in order to control tumorous growths. Holman feels that the only effective way to destroy the malignant cells which are already deficient in catalase and sensitive to overoxidation is to keep up a continued administration of an active oxidizing agent. Hydrogen peroxide is the oxidizing agent of choice; it is an excellent ionizing solvent and is obviously of great importance in most living cells.

Loiseleur[34] has pursued a series of experiments in which he demonstrates that the effects of irradiation result from primary radioactivation of the molecular oxygen found in solution in the irradiated tissues.

Horgan and associates[35] showed two types of reactions with various organic peroxides. On the basis of total peroxide content, auto-oxidized linoleic acid was more toxic by intraperitoneal injection than any of the simple peroxides. They found evidence consistent with the view that radiation toxicity is due to initiation of chain auto-oxidation of essential fatty acids producing lethal doses of peroxides.

Andersen[36] showed that cells are more sensitive to irradiation during mitosis. The most sensitive stage of mitosis appears to be prophase or late premitotic phase. If cells are irradiated at this time, they show a delay or inhibition of mitosis.

Mitotic Index.—In the normal specimens the mitotic index of the younger age group was approximately doubled in the older age group. The increase was from 0.79 to 1.69. Marwah[1] states that this finding would seem to contradict the common concept of aging, according to which bodily processes slow down and the regenerative capacity of the tissues diminishes with age.

When normal tissue of a similar age range was subjected to 30 per cent hydrogen peroxide for a period of thirty days, the mitotic index of both age groups was increased. The younger age group had an eightfold increase in its mitotic index, going from 0.79 to 6.50. The older age group had a fivefold increase, going from 1.69 to 8.80. From these figures, we can see that the older age group did not show the same proportional response as the younger age group.

If our hypothesis, as stated in the initial discussion, is true with regard to a colchicine-like action of hydrogen peroxide, then the above findings would seem to contradict Marwah's previous statement. Since the increase in the mitotic index was, in proportion, greater in the young subjects than in the older ones, this would seem to indicate a greater number of cells entering mitosis in the young or a less damaging effect of hydrogen peroxide in the older men.

From this colchicine-like effect we can also see that the mitotic index, as a measuring device, is only a relative mechanism. Therefore, we feel that the mitotic index as a tool is only as important as the set of circumstances under which it is used and evaluated.

Cell Density.—The density of the cell population of the older age group as compared with the younger age group was slightly greater in both the control and experimental specimens. When the control and experimental specimens were compared, the over-all cell density in the latter group was seen to decline. The experimental group's cell-density decrease could be attributed to a larger size of the cells and/or to a greater intercellular space. Because of the irregular shape of the prickle cells, even mere estimates of size would be a rather formidable task. The basal cells were seen to increase in size in the experimental group. Therefore, this could account for the over-all decreased cell density and possibly the apparent decreased reproductive capacity of the experimental cells.

The above statements could once again support the hypothesis that a potential poisoning of cells is occurring. The decreased number of cells and the increased size of the cells are more evidence to reaffirm the initial premise of a colchicine-like action occurring from hydrogen peroxide.

Significance of Differential Counts of Phases in Mitosis.—The incidence of the various phases of mitosis were somewhat altered with the differences in age and specimens. The most obvious alteration was in the greater incidence of prophase in experimental specimens from both the younger and older age groups.

This finding could add further impetus to the colchicine-like action of hydrogen peroxide. The prolonged exposure to hydrogen peroxide could be retarding or "holding" the cells undergoing mitosis in prophase.

Location of Mitosis and Plane Division.—Mitoses occur mainly in the basal layer and in the layer immediately overlying it, or the suprabasal area. Very few mitoses were located in the higher strata of the rete ridges.

The waviness of the junction of the lamina propria and epithelium makes it difficult to be sure whether a given nucleus is in the basal or suprabasal layer. If a rete ridge is cut tangentially, cells which actually are in the basal layer may appear to be in much higher strata of the rete ridge. This error can be avoided only by comparing serial sections.

Approximately 50 per cent of mitoses were in the basal layer. This is similar to the results obtained with human skin as reported by Pinkus[37] and Rothman.[25] The oxygenated specimens had a slightly higher number of mitoses in the basal layer. However, the results were reasonably the same for the two variables. This result did vary a great deal from that reported by Marwah,[1] who found 23 per cent of mitoses located in the basal layer and 47 per cent in the prickle layer of human attached gingiva.

In ascertaining the plane of cell division, we used only cells in anaphase and telophase. Many times it is difficult to determine the plane of cell division in prophase and metaphase; therefore, these phases were eliminated. Approximately less than two-thirds of the cells were found to be dividing at a plane perpendicular to the basement membrane. Because so many cells were found in a relationship to the basal layer, it can definitely be said that the basal layer is one of the contributing factors to mitosis in the suprabasal layer. It seems

obvious that many of the daughter cells must have ascended into the suprabasal layer and may or may not have undergone further multiplication. This may give further light to Cowdry's[38] concept of vegetative and differentiating intermitotics.

The vast similarity of planes of division in the control and experimental groups may once again indicate a potential "holding" mechanism being placed on the cells by the hydrogen peroxide.

SUMMARY AND CONCLUSIONS

The purpose of this investigation was to study the mitotic activity of normal human oral epithelium and, in addition, the possible alterations in mitosis patterns when normal epithelium is exposed to hydrogen peroxide.

1. The epithelium of human gingiva was studied and mitotic indices were established. In order to establish a control, we studied the normal gingivae of subjects from two different age ranges—a younger age group (average age, 25 years) and an older age group (average age, 56 years). The mitotic index in the control specimens was 0.79 for the younger age group and 1.69 for the older age group.

2. The mitotic index was greatly altered upon exposing the two age groups to 30 per cent hydrogen peroxide for thirty days. The mitotic index in the experimental younger age group increased eightfold, from 0.79 to 6.50. In the older age group it increased fivefold, from 1.69 to 8.80.

3. The over-all cell density of the experimental group was decreased when compared with that of the control group. In both groups, however, the older age group had a greater density than the younger age group.

4. After single exposures of 30 per cent hydrogen peroxide for a thirty-day period, an apparent prolongation of the mitotic period occurred. This seemed to be more specific in prophase. This apparently gives evidence that the mitotic process is retarded by hydrogen peroxide. Thus, the apparent increase in mitotic index was not a true reflection of the mitotic activity. Therefore, mitotic index is only relative to the set of circumstances under which it is used and evaluated.

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Section by the

ARMED FORCES AND PUBLIC HEALTH SERVICE

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BONE–SOFT-TISSUE RELATION IN LATERAL JAW ROENTGENOGRAMS

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OPERATIONS for the surgical correction of malocclusion, whether the jaws are prognathic or retrognathic, result in soft-tissue alteration. The use of a radiopaque paste shows soft-tissue contour and bone relation on the same

Fig. 1.—Radiopaque paste being mixed.

film. This method provides an excellent basis for preoperative planning and the maintenance of records, as well as accurate postoperative comparison of surgical results.

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Fig. 2.—Paste applied on the patient.

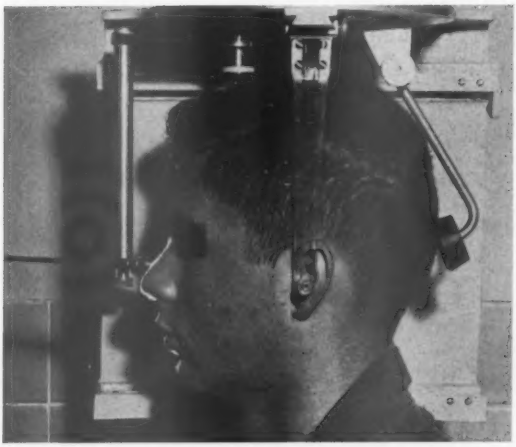

Fig. 3.—Patient in position for cephalometric studies.

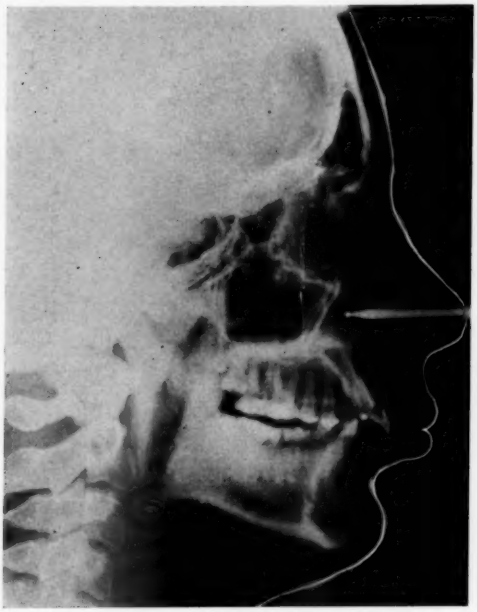

Fig. 4.—Preoperative view of retrognathic patient.

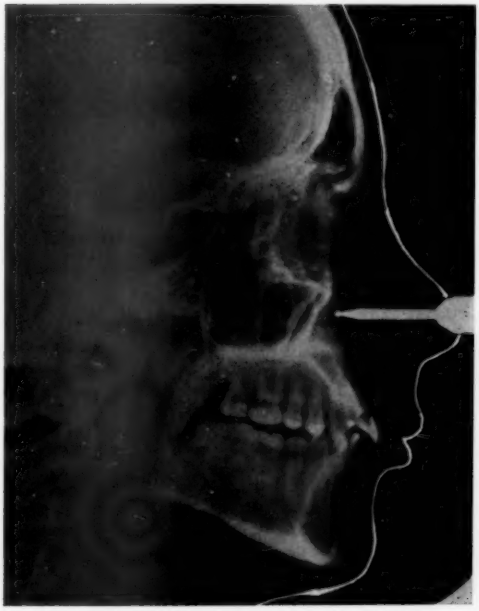

Fig. 5.—Postoperative view of retrognathic patient.

Fig. 6.—Preoperative view of prognathic patient.

Fig. 7.—Postoperative view of prognathic patient.

Barium sulfate powder is mixed with ordinary tap water, creating a thin paste. A camel's-hair brush is used to apply the paste to the soft-tissue contour in the profile position. The desired x-ray films are then taken, furnishing a complete lateral study. The paste is completely nontoxic and can be removed easily.

The material is equally useful for furnishing a radiopaque landmark on the skin for relative orientation of other objects, such as foreign bodies, etc.

THE FACTORS OF AGE AND TOOTH SIZE IN HUMAN PULPAL REACTIONS

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IN A previous study,[1] an inverse correlation was found to exist between the incidence of inflammatory responses in the pulpal tissues resulting from operative procedures and the thickness of remaining dentine between the cavity floor and the pulp. Teeth of different morphologic types were grouped together to gain an adequate number of specimens for the study. This seemed permissible since the relationship of dentine to pulpal tissues was considered so basic that little variation between the specimens was expected, despite gross anatomic differences. It was suggested, however, that the results might be more meaningful if applied to larger individual groups of incisors, cuspids, premolars, or molars.

Criticism has often been directed at pulpal studies utilizing human teeth from persons of a wide age distribution, since several investigations have indicated that teeth uninvolved by caries, abrasion, or erosion become harder, sclerotic, and less permeable with increasing age.[2-5] These changes, which could increase the resistance of the tooth and pulp to future episodes of disease, could also modify pulpal reactions to cavity preparations. Zander[6] demonstrated that the pulps of older teeth reacted less severely to silicate cements than did those of younger teeth. For these reasons, Shroff[7] recommended using teeth from patients within a certain age group (10 to 15 years) who were free from general and local disease and not subject to abnormal functional stresses.

Willis and Worner[8] found that increases in pulpal temperatures were greater in anterior teeth than in posterior teeth. This raised the question of whether tooth size or the closeness of the pulpal tissues to the tooth surface in anterior teeth was the explanation for this fact.

It would be very difficult to obtain adequate human material if one confined his specimens to a certain restricted age, morphologic tooth type, and a standard thickness of remaining dentine between the floor of the cavity and the pulp tissue. Appreciating these facts, investigators are reluctant to discard any specimen because of variation in tooth size and patient's age. Although

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such conditions are more readily met in animal experiments, Shroff[7] has pointed out that they are of restricted value and must always be followed by confirmation on the human subject.

Stanley and Swerdlow,[9] when comparing various operative techniques, minimized the age factor by not permitting the average age of the patients in any one operative category to vary from the average age of the patients in the other categories by more than five years. To account for size factors, teeth were classified according to the increasing average diameter of the crown in a labiolingual or buccolingual direction as follows: (1) maxillary lateral and mandibular central incisors; (2) maxillary central and mandibular lateral incisors; (3) all cuspids; (4) all premolars; and (5) all molars.[10]

The eventual accumulation of sufficient numbers of teeth prepared by various operative techniques has now permitted the present study to be undertaken in an effort to determine what effects age and tooth size might have on the pulpal response.

MATERIALS AND METHODS

The teeth* were selected and paired from the following low-speed operative techniques:

Speed (r.p.m.)	*Cutting Instrument (Inverted Cone)*	*Coolant*
6,000	No. 37 diamond stone	Air-water spray
6,000	No. 37 diamond stone	None
6,000	No. 35 carbide burr	Air-water spray
6,000	No. 35 carbide burr	None
20,000	No. 37 diamond stone	Air-water spray
20,000	No. 37 diamond stone	None
20,000	No. 35 carbide burr	Air-water spray
20,000	No. 35 carbide burr	None

Teeth prepared by these techniques were used because the pulpal responses to high-speed techniques were generally too minimal to permit contrasting differences. Typical Class V cavity preparations had been cut in the gingival regions of the facial surfaces of all the teeth and filled with zinc-oxide and eugenol prior to extraction at various time intervals.

To test the age factor, a group of younger teeth were paired with older teeth when a difference in age exceeded ten years. Both members of a pair came from the same operative category, were of similar tooth size, and had gone through similar postoperative extraction periods. The patients ranged in age from 23 to 58 years. The difference in age ranged from eleven to thirty-one years, with an average age difference of 17.8 years.

To test the factor of tooth size, smaller teeth were paired with larger teeth when a difference of at least two tooth sizes existed according to the tooth-size

*All the specimens were derived from previously published studies conducted in an effort[1, 9] to determine the relative traumatic capacity of various operative speeds and techniques.

classification given above. As above, both members of a pair came from the same operative category and had gone through similar postoperative extraction periods.

The teeth were also matched with respect to the thickness of the remaining dentine (R.D.) between the floor of the cavity preparation and the pulp. The difference in thickness of the remaining dentine between the two teeth of a pair was not permitted to exceed 0.2 mm. Although the pulpal response generally becomes more severe as the cavity preparation approaches the pulp chamber, it was felt that a difference in R.D. thickness of this magnitude was too slight to afford any biologic advantage to either member of a pair.

If age or tooth size could provide any protection to the pulp with the above-mentioned factors being kept as nearly equal as possible, one could expect to find a greater number of milder lesions in the older or larger teeth. The pairs were then appraised microscopically, and the lesion found in the older or larger tooth was recorded as being more severe or less severe than its younger or smaller counterpart. Histopathologic comparisons were made by noting the size of the lesion, the incidence and prominence of the cellular infiltrations, cellular displacement, and irregular dentine formation.

RESULTS

In the age factor group, eleven older teeth (45.8 per cent) showed a more severe lesion and thirteen (54.1 per cent) demonstrated less severe lesions than their younger counterparts. In the older teeth where the pulpal horns or occlusal portions of the coronal pulp chamber were filled in with irregular dentine, the lesions were confined to the cut tubules not lined by the irregular dentine. The presence of the irregular dentine modified only the size and not the intensity of the lesion.

TABLE I. FACTOR OF REMAINING DENTINE THICKNESS (R.D.) WHEN 0.2 MM. OR LESS

	NUMBER OF ACTUAL LESIONS	NUMBER OF CORRECT PREDICTIONS
Age group	24 (13 less severe; 11 more severe)	16 (9 less severe; 7 more severe)
Tooth-size group	49 (21 less severe; 28 more severe)	18 (6 less severe; 12 more severe)
Totals	73	34

In the tooth-size factor group, twenty-eight larger teeth (57.1 per cent) showed more severe lesions and twenty-one (42.9 per cent) showed less severe reactions.

Except possibly for a slight increase in the number of more severe lesions in the larger teeth, neither age nor tooth size appeared to influence the type of reaction obtained. Consequently, a determination of how accurate a pre-

diction of degree of response could be made on the basis of a difference in remaining dentine thickness no greater than 0.2 mm. was decided upon. Before the microscopic comparisons were made, a predicted number of more severe and less severe lesions to be found in the older and larger teeth was recorded (Table I). These predictions were determined by the R.D. value of the older and larger tooth. For example, if the older or larger tooth possessed an R.D. value greater than its counterpart, a less severe lesion was predicted, and vice versa.

When the predicted lesions were tallied against the type of lesions actually found, the following results were obtained (Table I): Sixteen correct predictions were made in the age factor group and eighteen were made in the tooth-size factor group. Altogether, thirty-four (46.6 per cent) correct and thirty-nine (53.4 per cent) incorrect predictions were made, a discrepancy of more than 50 per cent. When the R.D. difference is 0.2 mm. or less, it is impossible to predict which teeth will reveal the more severe pulpal reactions.

DISCUSSION

Age, as manifested in the form of tooth hardness, sclerosis, or decreased permeability, provided no obvious protection to the pulpal tissues subjected to cavity preparation, except in the older teeth where the pulpal horns or occlusal portions of the pulpal chambers were filled in with irregular dentine. This characteristic affected only the size of the lesions; it did not modify their intensity. Apparently, one need not be concerned with variations in age of the patients from whom the specimens came so long as irregular dentine does not underline the tubules being cut in the operative procedure. The age span between the youngest members and oldest members of the study was too short (the maximum age difference was thirty-one years) to permit the demonstration of any pulpal protection possibly due to maturation sclerosis. If age does play a part, its contribution is too slight to be recognized in the light of knowledge concerning the overwhelming effect of remaining dentine thickness.

TABLE II

	PAIRS OF TEETH	GROUPS OF TEETH	AVERAGE POSTOPERATIVE PERIOD (DAYS)	AVERAGE PREPARATION TIME (SECONDS)	AVERAGE AGE (YEARS)
Age factor	24	Younger	34.7	41.9	29.3
		Older	39.9	39.3	47.1
Tooth-size factor	49	Smaller	26.1	36.8	41.3
		Larger	26.2	39.4	40.9

Differences in age and preparation time (Table II) were too slight to contribute to the slightly higher incidence of more severe responses in the larger teeth. The average age difference between the smaller and larger teeth was less than one year. The average difference in preparation time was less than three seconds.

Experience has taught us that lesions involving the uppermost portion of the pulpal horns, where the depth or breadth of the pulpal tissues is very thin,

require a more prolonged resolution period. The distal position of the horns in relation to the main blood supply evidently contributes to this circumstance. Because of the large number of more severe lesions in the larger teeth, all the larger teeth were re-examined for pulpal horn involvement even though typical Class V cavity preparations had been made toward the cervical area. More severe lesions occurred in twenty-eight larger teeth, but only three showed pulpal horn involvement. Another explanation of the incidence of more severe lesions in larger teeth must be forthcoming.

SUMMARY

This study was undertaken to determine what effect age and tooth size might have on the pulpal response of teeth subjected to the trauma of cavity preparation.

Age, in itself, provided no obvious protection except by the presence of irregular dentine in the pulpal horns or occlusal portions of the pulp chambers. Pulpal lesions did not occur where irregular dentine lined the cut tubules prior to cavity preparation. When irregular dentine was not found in the older teeth, however, the intensity of the lesions was not modified.

Tooth size did not appear to play an important part in determining the pulpal response. However, since a slight increase in the number of more severe lesions did occur in the larger teeth, despite similar thicknesses of remaining dentine, it is recommended that in future studies a comparable average tooth size for each group of teeth being studied be maintained in order to minimize the bias of the results. An adequate explanation for the slightly higher incidence of more severe lesions in the larger teeth is still needed.

Closeness of cavity preparation to the pulp (remaining dentine thickness) when teeth with R.D.'s differing by more than 0.2 mm. are being compared remains the most useful factor in the understanding and interpretation of the responses of the human dental pulp to known traumatic stimuli.

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PERIAPICAL CEMENTOBLASTOMA

Report of a Case

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PERIAPICAL roentgenolucencies, when observed in routine roentgenograms, often present great diagnostic problems. They may represent cysts, neoplasms, or cementoblastomas. These cementoblastomas are made up of connective tissue, generally of limited growth, and may be spoken of as cementifying fibromas. The lesions originate in the periapical region of the normal permanent dentition as a result of proliferation of cells of the periodontal membrane. The cementoblastoma often simulates in the x-ray film a dental granuloma or small radicular cyst, and it is mistakenly diagnosed as a calcified granuloma if cementum has been deposited in the lesion.

There are many instances in which the lesion remains very small and the roentgenopacity varies so little from the surrounding bony process that roentgenographic diagnosis is uncertain. Differential diagnosis depends largely upon the stage of development at which the periapical condition is observed and upon the clinical appearance of the involved teeth in association with pertinent history.

Roentgenographically, one may distinguish several stages of development. The osteolytic stage, which represents the immature cementoma, is composed of cellular tissue with no calcified structure deposited as yet. The tumor forms at the expense of bone, and loss of the bone produces a roentgenolucency which is seen as a dark area in the x-ray film. In the cementoblastic stage the tumor has started to form cementum. The deposited tissue which becomes calcified is roentgenopaque and, therefore, produces an image on the x-ray negative. Occasionally, both stages of development may be seen to be going on at the same time.

The presence of roentgenolucent areas around the apex of a tooth which has normal color, vitality, and no history of trauma, pain, or pulpitis should suggest this growth to be a cementoblastoma. This is particularly true if the tooth involved is a mandibular incisor (Fig. 1).

CASE REPORT

The case illustrated in Figs. 1, 2, and 3 is that of a 38-year-old white woman with no pain symptoms. The teeth were normal as to color and vitality. There were no systemic complaints.

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Fig. 1.—Apical roentgenolucency of mandibular incisor. Note absence of any evidence of caries. (Osteolytic stage.)

Fig. 2.—Mandibular incisor areas showing roentgenopacity of tumor. (Cementoblastic stage.)

The case is illustrative of several stages of development of the tumor, since both roentgenolucent areas (osteolytic stage) and cementicles (cementoblastic stage) are shown (Figs. 2 and 3).

Fig. 3.—Mandibular incisor areas showing roentgenopacity of tumor. (Cementoblastic stage.)

SUMMARY AND CONCLUSIONS

Periapical roentgenolucent areas in man are not an uncommon finding. The significance of the lesion lies in the fact that it should be included in the differential diagnosis of apical pathology.

As this tumor is slow growing, surgical treatment becomes a matter of choice. If the lesion is small and its removal would require devitalization or extraction of normal useful teeth, it should be kept under observation for an indefinite period of time. If the tumor is large, containing a great deal of fibrous tissue, operation is advised, particularly if expansion of the bone and facial disfigurement have occurred.

ANNOUNCEMENTS

American Academy of Dental Medicine

The Academy will hold a cruise convention aboard the luxury liner *S. S. Ocean Monarch* to Bermuda and Nassau over the Memorial Day week end, May 26 to June 3, 1961, combining a comprehensive scientific session with a program of recreation.

The theme of the scientific program is "Recent Trends in Dental Medicine." Lectures will be given by Hermann Becks, Professor of Oral Biology, University of California School of Dentistry; J. Lewis Blass, Professor of Periodontia and Oral Medicine, New York University College of Dentistry; and Milton Hyman, Professor of Oral Diagnosis and Roentgenology, New York University College of Dentistry. Two symposiums will be conducted by members of the various sections of the Academy.

While in Bermuda members of the Academy will be guests of the Bermuda Dental Society at a buffet dinner–lecture. Dr. Hermann Becks, President of the Academy, will address the joint meeting.

Superb service and cuisine, nightly dancing and entertainment, sight-seeing, and sports activities will be included in the recreational program.

All interested dentists and physicians, as well as Academy members and their families, are welcome. For details and reservations, address Dr. Howard Ward, General Chairman, 15 Bond St., Great Neck, New York.

Twelfth Annual Berkshire Conference in Periodontology and Oral Pathology

Major General Joseph L. Bernier, Assistant Surgeon General and Chief of the Dental Corps, United States Army, will be one of the principal speakers at the Conference, which will be conducted at Eastover in Lenox, Massachusetts, June 18 to 22, 1961. Other lecturers on the Conference faculty will be Finn Brudevold, Irving Glickman, Reidar F. Sognnaes, John B. MacDonald, Sigurd Ramfjord, Everett Shapiro, David Weisberger, and Frank M. Wentz.

The Conference is open to general practitioners and dentists in specialized practice. Information may be obtained from Tufts Berkshire Conference, Tufts University School of Dental Medicine, 136 Harrison Ave., Boston 11, Massachusetts.

Dental Internship Available at Hospital for Joint Diseases

The hospital announces that a position is available for a rotating dental intern in its approved Department of Dentistry for the year beginning July 1, 1961. The department is headed by Dr. Stanley J. Behrman, Attending in Dentistry, and the training includes oral surgery, orthodontics, oral pathology, radiology, and periodontics.

The hospital provides its interns with room and uniforms, plus a cash stipend of $110.00 a month and insurance coverage.

Applications should be addressed to Executive Director, Hospital for Joint Diseases, 1919 Madison Ave., New York 35, New York.

Oral Surgery Internship Available at Mount Sinai Hospital

A one-year approved oral surgery internship is available from July 1, 1961, to June 30, 1962. Applicants are instructed to address Dr. A. S. Nathan, Director, Department of Dental and Oral Surgery, The Mount Sinai Hospital, Fifth Ave. and 100th St., New York 29, New York.

Boston University School of Medicine

The Oral Surgery Section offers a one-year oral surgery course planned to meet the requirements of the American Board of Oral Surgery. It provides a foundation for clinical training, such as clinical internship and residency, leading to qualification for the practice of oral surgery as a specialty. This course, given annually, is designed to give, by lecture, clinical demonstration, operative clinic, and laboratory exercise, a systematic review of the clinical phases of oral surgery. This is coordinated and correlated with the pertinent preclinical sciences. The fundamental principles of diagnosis and treatment of the various manifestations in the field of oral surgery are covered, with specific emphasis on etiology and pathology. Instruction is given in oral surgery, principles of general surgery, animal surgery, radiology, general and oral pathology, oral medicine, physical diagnosis, medicine, dermatology, clinical laboratory procedures, anesthesia, physiology, biochemistry, pharmacology, anatomy (head and neck), bacteriology, surgical prosthesis, and oncology. The course is under the direction of Dr. Kurt H. Thoma, Professor of Oral Surgery.

The Orthodontics Section offers a course of two academic years intended for those who wish to prepare themselves for the practice of orthodontics as a specialty. This course consists of lectures, clinical demonstrations, clinics, and laboratory exercises covering the various phases of the specialty. Courses are given in the preclinical sciences pertaining to this field. Applications are being accepted for the program which begins in the fall of 1962. The course is under the direction of Dr. Herbert I. Margolis, Professor of Orthodontics.

The Prosthetics Section offers a one- or two-year course in general prosthetic dentistry. It is intended for those who wish to prepare themselves for the practice of prosthodontics as a specialty and/or who may wish to prepare for the specialty board examinations. This course consists of lectures, clinical demonstrations, operative clinics, and laboratory exercises in all clinical phases of the field. These are coordinated and correlated with pertinent preclinical sciences. The course is under the direction of Dr. Leo Talkov, Assistant Professor of Prosthetic Dentistry.

The Endodontics Section offers a one-year course to dentists wishing to specialize in the field of endodontics. This course consists of lectures, clinical demonstrations, clinics, and laboratory exercises covering the various phases of this specialty. Instruction is given in the preclinical sciences pertaining to this field. The course is under the direction of Dr. Herbert Schilder, Assistant Professor of Endodontics.

The Oral Pathology Section offers a two-year course intended to prepare dentists for teaching or research in this field. The program includes the pertinent basic sciences and clinical oral medicine. The related curriculum consists of anatomy, biochemistry, physiology, microbiology, oncology, dermatology, general pathology, and oral pathology. The general pathology is given in connection with the Pathology Department of the Massachusetts Memorial Hospitals.

For application or information, please apply to Department of Stomatology, Boston University School of Medicine, 80 East Concord St., Boston 18, Massachusetts.

New York University College of Dentistry

The Department of Periodontia announces that a one-day basic course in the uses of ultrasonics in periodontal therapy will be given on Friday, April 14, 1961, from 9:30 A.M. to 5:30 P.M.

The course, designed to acquaint the general practitioner with the scientific principles and clinical uses of ultrasonic vibrations in periodontal therapy, will be directed by Dr. Sidney Sorrin, assisted by Drs. N. Alderman, A. Arvins, S. J. Ewen, M. J. Pomeranz, M. Simring, and S. S. Stahl.

Applications for the course may be obtained by writing to Director, Post-Graduate Division, New York University College of Dentistry, 421 First Ave., New York 10, New York.

Traineeships at University of Pennsylvania

Research and teaching traineeships in the basic sciences, in periodontology, and in the comprehensive treatment and rehabilitation of the cleft palate patient are available at the School of Dentistry, University of Pennsylvania. All traineeships are supported by the United States Public Health Service. Annual stipends vary from $4,500.00 and up, depending on the qualifications and needs of the successful candidates.

Inquiries concerning the basic science and cleft palate traineeships should be sent to the Chairman, Committee on Traineeships and Fellowships, University of Pennsylvania, School of Dentistry, 4001 Spruce St., Philadelphia 4, Pennsylvania. Inquiries for the traineeships in periodontology should be sent to: Dean, Graduate School of Medicine, 237 Medical Laboratories, University of Pennsylvania, Philadelphia 4, Pennsylvania.

Tufts University School of Dental Medicine

A special participation course in periodontology (DPG 605) will be given Monday through Friday, Oct. 9 to 13, 1961, by Dr. Irving Glickman and staff. Tuition for this course will be $175.00.

Further information may be obtained by writing Director of Division of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine, 136 Harrison Ave., Boston 11, Massachusetts.

Albert Einstein College of Medicine
Yeshiva University

The Department of Dentistry announces the following postgraduate courses:

Anesthesiology DPD 11—Pre-medication, Co-medication, and Supportive Therapy in Dental Practice. April 6 and 7, 1961. Dr. Stanley R. Spiro and associates. $60.00.

Anesthesiology DPD 19—Preoperative and Pre-Anesthetic Evaluation Techniques for All Dental Patients. June 9, 1961. Dr. Stanley R. Spiro and associates. $30.00.

Dento-Alveolar Surgery DPD 33. May 4 and 5, 1961. Drs. Herbert Calman, Stanley J. Behrman, William Rakower, and associates. $50.00.

Advanced Oral Pathology DPD 29. Three consecutive days, December, 1961. Dr. Ernest Baden and associates. $75.00.

Periodontics DPD 69. Oct. 30 to Nov. 3, 1961. Drs. Irving Yudkoff, Marvin N. Okun, and associates. $175.00.

For further information and application blanks about specific postgraduate courses, write to Director, Postgraduate Dental Division, Albert Einstein College of Medicine, Yeshiva University, Eastchester Rd. and Morris Park Ave., Bronx 61, New York.

West Virginia University

The School of Dentistry, which is introducing its first series of postgraduate refresher courses this year, announces that the following courses will be presented:

P.G. 11. Practical Periodontics for the General Practitioner. April 7 and 8, 1961. C. M. Fraleigh.

P.G. 1. Refresher Course in Crown and Bridge Prosthodontics. April 10 and 11, 1961. J. F. Johnston and J. D. Adams.

P.G. 5. High Speed Instrumentation. April 14 and 15, 1961. R. E. Sausen and W. F. Braasch.

P.G. 12. Complete Denture Impressions and Jaw Relationship Records. April 17 to 19, 1961. J. B. Davis and J. D. Eselman.

P.G. 2. Refresher Course in Principles and Technics of Crowns and Bridges. One day a week, April 27 and May 4, 11, 18, and 25, 1961. J. D. Adams.

P.G. 8. Cephalometry. May 22 to 24, 1961. V. Sassouni and W. M. Krogman.

P.G. 6. Inlay Restorations. One day a week, June 16, 23, and 30, 1961. R. E. Sausen.

Further information may be obtained from Dr. Kenneth V. Randolph, Dean of the School of Dentistry, West Virginia University, Morgantown, West Virginia.

University of Pennsylvania

The School of Dentistry announces the following postgraduate courses:

Complete Denture Prosthesis. April 17 to 21, 1961. M. M. DeVan and associates.

Function—Prime Object of Dentistry. April 24 to 28, 1961. Arne G. Lauritzen.

Extraoral Tracers in Rehabilitation of Function. May 1 to 4, 1961. Ernest R. Granger and William Maxwell.

Principles of Occlusion. May 3 to 5, 1961. Lewis Fox and associates.

Biologic Aspects of Endodontics. May 22 to 26, 1961. Samuel Seltzer and I. B. Bender.

For further information, please write Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

University of North Carolina

The School of Dentistry announces the following refresher courses:

Clinical Periodontia and Oral Pathology. June 6 to 10, 1961.

Dentistry for Children, I, II, and III. July 10 to 14, 17 to 21, and 24 to 28, 1961.

Design and Case Analysis for Clasp Partial Dentures. June 15 to 17, 1961.

Further information may be obtained from the Department of Postgraduate Dental Instruction, University of North Carolina School of Dentistry, Box 750, Chapel Hill, North Carolina.

Balint Orban Memorial Seminar

The Seminar (formerly known as the Rocky Mountain Dental Seminar) will be held in Boulder, Colorado, June 25 to 28, 1961. The theme for the Seminar is "Clinical and Microscopic Pathology of the Periodontium and Other Oral Structures." The outstanding group of essayists includes Drs. S. N. Bhaskar, Henry Goldman, Maynard K. Hine, and Harry Sicher.

Further details and application may be obtained by writing to Mrs. James Manella, Secretary, Balint Orban Memorial Seminar, 1700 Mesa Rd., Colorado Springs, Colorado.

New York University College of Dentistry

A Workshop in Practice Management, Doctor-Patient Relations, and Motivation of Patients will be conducted by J. Lewis Blass and associates during the week of June 19 to 23, 1961.

Applications should be made to Postgraduate Secretary, New York University College of Dentistry, 421 First Ave., New York 10, New York.

University of Oregon

A three-year graduate program in oral pathology is being offered at the University of Oregon Dental School. The first year of the program consists of resident-type training in general pathology at the University of Oregon Medical School. The other two years are taken under the direction of Drs. Norman H. Rickles and Robert J. Bruckner at the University of Oregon Dental School; during this period course work will be done in oral pathology, biochemistry, bacteriology, genetics, and statistics. Research activity leading to a thesis completes the requirements for the Master of Science degree in oral pathology. Financial support is available through the Teacher-Training Program of the United States Public Health Service.

All inquiries should be addressed to Dr. Ellis B. Jump, Chairman of the Graduate Committee, University of Oregon Dental School, 611 S. W. Campus Dr., Sam Jackson Park, Portland 1, Oregon.

REVIEWS OF THE LITERATURE

BOOK REVIEW

Partial Dentures. Merrill G. Swenson and Louis G. Terkla. Second edition, St. Louis, 1959, The C. V. Mosby Company. 387 pages; 403 illustrations, 6 in color; references, glossary, and index. Price, $10.50.

Partial Dentures, an exceptionally well-prepared, profusely illustrated text, will bring the practitioner and student up to date in the science of partial dentures. The text is presented in two parts; the first deals with the fundamentals and principles of the procedures and the second is concerned with technique. This is a complete reversal of the first edition, and a decided improvement.

Many excellent ideas are advanced on the bases of the authors' broad experience in the field of prosthetic dentistry. Part I contains a new, logical, and scientific approach to the classification of partially edentulous cases. It is hoped that the Academy of Prosthetic Dentistry will see fit to establish a new classification similar to the one advanced in this text. There is a chapter devoted entirely to the importance of centric relation and centric occlusion as determined by the terminal hinge position. Unfortunately, variances in terminology appear in Chapter 8. The authors refer to the once standard Kennedy bar and closed Kennedy bar as the continuous bar and the apron bar. The terms "apron bar," "Kennedy bar" and "closed Kennedy bar" do not appear in the glossary. In Chapter 8 it is noted the advantages are given for the continuous bar and the apron bar, but nothing is said about disadvantages or when the continuous bar should be used instead of the apron bar.

The glossary at the end of the text is an excellent contribution. Another new and useful idea appearing here is a list of references at the end of the text rather than after each chapter; however, the reference list would be easier to use if the subjects had been arranged alphabetically.

In the words of the authors, "without a common language, the advancement of knowledge . . . is slow." This text does a service to the student, the teacher, and the profession in helping to establish a common language in prosthetic dentistry. It may be used to great advantage by the practitioner and student alike.

H. H. Burkart.

ABSTRACTS OF CURRENT LITERATURE

Osteogenic Sarcoma of the Mandible. A. C. Gomez, R. D. Youmans, and R. G. Chambers. Am. J. Surg. **100:** 613, October, 1960.

Osteogenic sarcoma of the mandible is usually grouped along with tumors of other osseous structures in surveys of bone tumors presented by orthopedic surgeons and pathologists.

Clinically, it is frequently confused with benign tumors. Two cases are reported. The first involved a 32-year-old white woman whose chief complaint was recurrent tenderness and swelling of the left mandibular cuspid area of two months' duration. Excisional biopsy was reported on microscopic examination as ossifying fibrous dysplasia or a possible osteogenic sarcoma. The lesion recurred in six weeks, and the diagnosis of osteogenic carcinoma was made on a second biopsy. Treatment following the diagnosis of osteogenic sarcoma included irradiation followed by surgery, which included a left hemimandibulectomy. There was no evidence of active disease thirteen months after surgery.

The second case was that of an 18-year-old white girl who had had a painful swelling in the right mandibular region for four weeks. Following removal of the second premolar, the swelling increased. An excisional biopsy was done four weeks after the onset of symptoms, and the diagnosis of osteogenic sarcoma was made. Treatment included irradiation followed by surgery. Nine months after treatment no evidence of active disease was found.

The authors feel that early diagnosis and an aggressive approach in therapy takes this disease out of the hopeless category.

T. J. C.

Lymphangioma of the Tongue. H. Mason Morfit. A. M. A. Arch. Surg. 81: 761, November, 1960.

Lymphangiomas are tumors composed of true lymph channels and cystic dilatations. This article reports efforts to treat eight patients with lymphangioma of the tongue, three of whom were followed for ten to fifteen years. In all eight patients, the tumors were noted at or shortly after birth. They were grapelike excrescences on the surface. The tongue protruded from the mouth as it increased in size, and the teeth were forced out of occlusion.

Surgery is the treatment of choice. The author cites the importance of marking the lines of excision on the tongue prior to making the incision.

The procedure is described in detail and the article is recommended to the oral surgeon.

T. J. C.

Carcinoma of the Palate. E. P. Sherokov. Am. J. Surg. 100: 530, October, 1960.

Thirteen Panamanian women with epidermoid carcinoma of the palate were seen. All of these women had, for years, smoked rolled tobacco leaves of small cigars with the lit end within the mouth and against the mucosa of the hard palate. Local pain was the most common symptom. A foul discharge was seen when the mucosal surface was broken and infection occurred.

Surgical extirpation is the method of choice. The cheek is reflected by cutting through the lower lip in the midline and extending the incision below the mandible. The mandibular attachments are freed, which gives excellent exposure. The diseased part is on the surface. The neck dissection precedes the intraoral work. Although surgical excision of these tumors is considered relatively simple, reconstruction of the defect is recognized as a problem.

T. J. C.